Community
Medicine
اللجنة العلمية 37A
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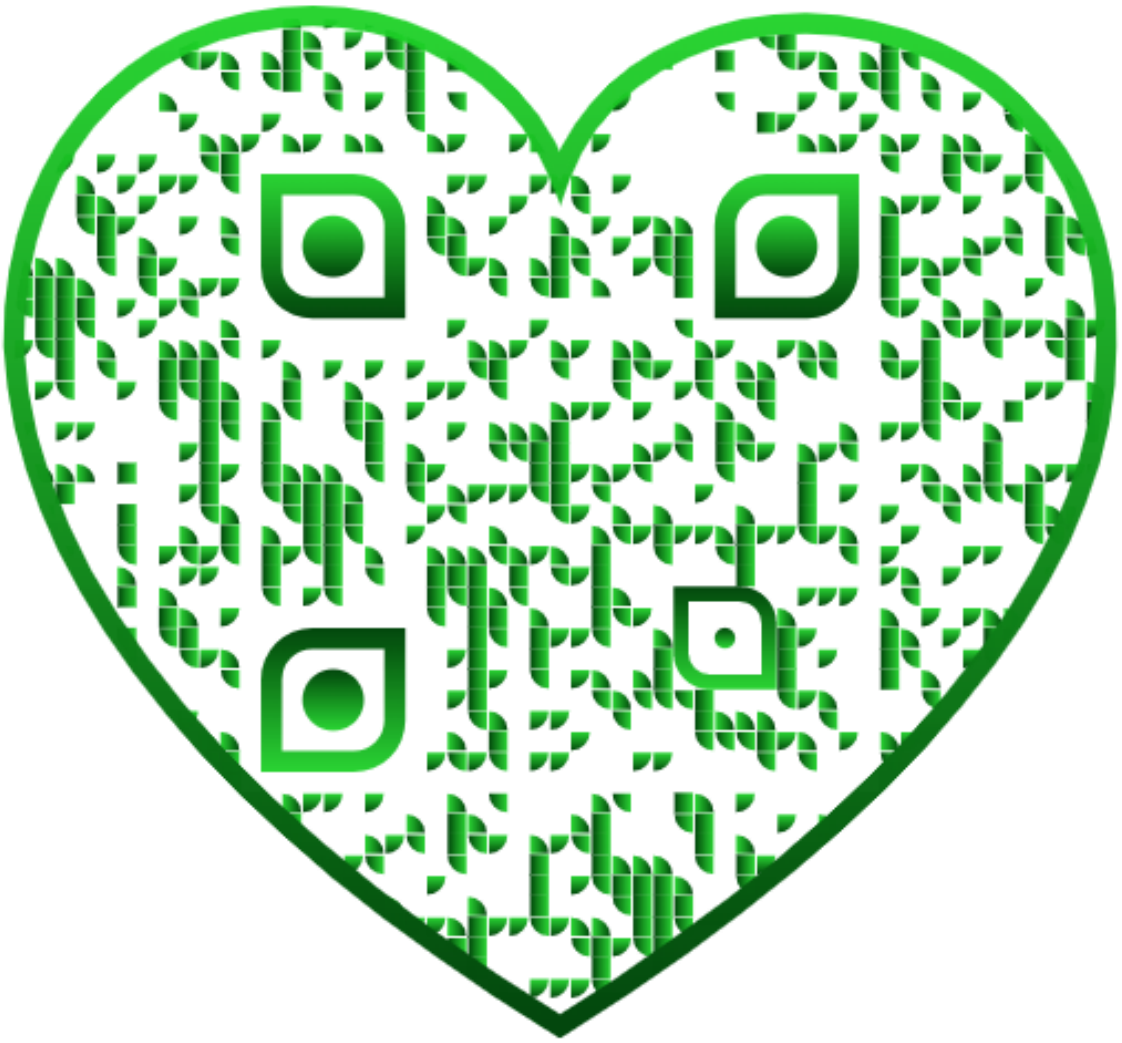

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Epidemiology and Control of Viral hepatitis

BY:-

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SANA’A - 2017

Definition of viral hepatitis

Viral hepatitis is an inflammation of the liver parenchymal hepatocytes due to viral infection which mainly caused by viruses A, B, C, D, E and G.

Viral hepatitis may presents without any clinical sings or symptoms (asymptomatic), with non –specific symptoms without jaundice (anicteric) or with symptoms that occur with jaundice (Icteric).

Hepatitis due to other viruses account for only 1-2% of cases (Cytomegalo virus, Epstein –Barr virus, Herpes simplex virus, Yellow fever virus and Ebola virus etc...)

Fulminant Hepatitis or Acute liver failure

It is a severe form of hepatic failure with extensive necrosis of the liver and encephalopathy within 8 weeks after the onset of acute liver diseases due to any cause.

About 70% caused by viral hepatitis, out of them 50% due to viral "B" other viral causes are: Hepatitis

A", E, D and "A on C"

Additional Causes are: Drugs, Toxins, shock, Wilson's disease, fatty liver of pregnancy and lymphomas

Study of Aetiology of Fulminant Hepatitis (%)

C-ry	HBV	HAV on CV	PA	T/D	HAV	Others
USA	15	39	18	15	8	5
UK	9	18	56	7	5	5
F-ce	46	19	2	20	4	9
Total	70	76	76	42	17	19

C-ry -country
F-ce -France
PA -Paracetamol
T/D -toxins/drugs

Clinical forms of viral hepatitis

Clinical forms can be divided into states which appear as follow:-

1. Carrier state
2. Asymptomatic infection
3. Acute hepatitis (symptomatic)
4. Chronic hepatitis and Cirrhosis
5. Fulminant hepatitis

Mode of Transmission

1. Blood and blood product mode Transmission (parenteral)
2. Sexual mode transmission
3. Contact mode transmission
4. Prenatal mode transmission
5. Others
6. Unknown

Phases of acute state

1. Incubation phase (period)
2. Asymptomatic pericteric phase(Prodromal)
3. Icteric phase
4. Convalescent phase

Classification of chronic hepatitis

Chronic hepatitis (CH) is defined as a status of vary symptoms from asymptomatic mild carriers to moderate and severe active hepatitis beyond 6 month and more.

1. Chronic persistent hepatitis (Mild and Moderate).

   Inflammation just between the portal area

   without destroying parenchyma architecture (compensate state).

   The laboratory manifestation Raise LFT with viral markers

2. Chronic severe active hepatitis (decompensate state)

   active Inflammation, Necrosis and fibrosis scattered

   throughout the lobular and cells of the liver

   (abnormal parenchyma architecture).

Causes of chronic hepatitis

- Viral: Hepatitis “B” + “D” and Hepatitis “C”
- Autoimmune Hepatitis
- Drugs: Methyldopa, Isoniazid, Ketoconazole, Nitrofurantion
- Hereditary: Alpha-1 antitrypsin deficiency, Wilson ‘s disease
- Others: Alcohol and Ulcerative Colitis

Diagnosis of Chronic Hepatitis

1. Clinical picture
2. Liver function test LFT, Viral markers (All viruses are RNA except “B” is DNA), and Auto antibodies
3. Alpha-fetoprotein
4. Liver biopsy – this procedure is useful in providing information about the extent of lever Damage
5. US and CT scan

Viral Hepatitis "A"

*The most common type of viral hepatitis particularly affects children and young adults, RNA virus, oral-fecal route transmission and arises from contaminated water, poor sanitation, overcrowding , milk and food

* Infected person:- Excretes virus in the feces for about 2 weeks before the onset of illness and for up 7days after

* Incubation period:- 2-6 weeks, no carriers, complete recovery, does not led to chronic and low mortality

* Diagnosis:- Epidemiological and clinical picture and by detect anti-HAV and RNA in the stool

*Course over 2-3 weeks, recovery by laboratory 9 weeks

*Treatment:- Bed rest, diet, I\V feeding and vitamins

Specific prevention:- a) Good Sanitation

b)Immunization by hepatitis "A" vaccine

(inj. Havrix) 0.5-1ml I\M

c) Passive immunization by immune globulin 0.6ml\kg I\

Viral Hepatitis “ E ”

Hepatitis “E” is RNA virus, water born transmission, similar to hepatitis “A”, But clinical illness more severe with mortality rate 1-2% compared with “A” 0.2%, It does not transform to chronic hepatitis.

* Diagnosis confirmed by Anti-HEV and HEV RNA in serum or in stool by PCR

* Prevention depends on good sanitation, No vaccine is available and immune globulins non effective

* Fulminant hepatitis rise to 20% in pregnant women, exact cause unknown

Viral Hepatitis “B” Definition

It is a common world- wide problem characterized by an inflammatory process of liver cells in acute form which can be develop to chronic case, cirrhoses and hepatocillular carcinoma (10-30 %), Fulminant hepatitis (1%), while 70 - 90% become asymptomatic carriers of HbsAg,

HbsAg carriers can develop “ later” to hepatitis which is depended on decreasing of immune system.

Virology

The Virus of hepatitis “B” is a member of Hepadana-viridae contain several antigens which can infected person and make immune response.

The genome of HBV is comprises capsule and core containing DNA and Polymerize enzyme.

The antigens of virus and their antibodies circulate in the blood where it can be very important in identifying “HBV” infection.

Genotypes of HBV

- Genetic classification of HBV has defined six genotypes A, B, C, D, E and F.
- B- and C- are predominant in the Far East.
- F- is predominant in Americas.
- E -is predominant in Sub-Saharan Africa
- A - and D- are more widely distributed.
- Also hepatitis" B" is classified into four major Subtypes (adw, ayw, adr, and ayr).

Epidemiology of viral hepatitis ”B”

I. Viral hepatitis “B” is a common world wide major health problem affecting 2 billions population (exposure).

II. The infection is endemic in most regions of the world with prevalence (carriers) of more than 300 million or about 5% of the world population.

III. Viral hepatitis “B” may occurs in 70-90% without symptoms

III.There are three levels of “HBV Prevalence” in the world :-

1. Highest rates

The prevalence of hepatitis “B” carriers (HbsAg) is 10% and more and anti-HBs is 80% and more.

(South East Asia, China, Equatorial Africa, Oceanic and South America).

2. Intermediate rates

The prevalence of hepatitis “B” carriers (HbsAg) is 5% and more and anti-HBs is 40% and more.

Eastern Europe, around the Mediterranean, South America and some of the Middle East Countries .

3. Lowest rates

The prevalence of hepatitis “B” carriers (HbsAg) is 0.1- 0.5 and more and anti-HBV 5% and more.

(Western Europe , North America and Australia)

Note:-

DNA - deoxyribonucleic acid is a genetic material of nearly all living organisms

RNA – ribonucleic acid occurring in the nucleus and cytoplasm of cells. In some viruses RNA is the genetic material

In Yemen

- Viral hepatitis is one of the priority health problem where it represent the one of the ten common endemic diseases that play a significant role in morbidity and mortality.

- The prevalence of HB carriers (HbsAg) is between 12% and 20% and some markers (anti-HBs) were found in 45 .5 % of study subjects.

- The carrier rate of HbsAg similar in all countries of the middle east.

Table3: Deferential Diagnosis of Viral Hepatitis " A&E " and "B & C ".

A and E	B and C
Viral hepatitis disease ➢ Infection agent:- ➢ 'A' and E virus RNA ➢ Occurrence:- ➢ World wide sporadic and epidemic with seasonal variation. ➢ Reservoir:- ➢ Man ➢ Mode of transmission:- ➢ Person to person by contact and fecal-oral route. ➢ Incubation period:- ➢ 15-45 days (30 days)	Viral hepatitis disease ➢ Infection agent:- ➢ 'B' virus DNA ➢ C virus RNA ➢ Occurrence:- ➢ World wide ,endemic with little seasonal variation. ➢ Reservoir:- ➢ Man ➢ Mode of transmission:- ➢ By the blood, saliva, semen and vaginal fluids. ➢ Incubation period for "B" ➢ 45-180 days (90 days). ➢ symptoms often arthralgia and

Clinical Picture of Acute Hepatitis “B” (more severe than all types of Hepatitis)

- 1. Incubation Period :-

  30 -180 mean 90 days

- 2. Pre-Icteric Period or

  Prodromal phase :-1-2 weeks

flulike syndrome, anorexia, nausea, vomiting, abdominal pain, and dark urine

- 3. Icteric period :- 1-3 weeks, jaundice, hepato- spleno - megaly, pale stool, dark urine, mild fever, Lymphadenopathy
- 4. Convalescent period- recovery phase:- to 3-6 weeks

HBsAg	Anti-HBs	Anti-HBc	HBeAg	Anti-Hbe	Interpretation
+	-	IgM	+	-	Acute HB
+	-	IgG	+	-	Chronic HB with active viral Replication
+	-	IgG	-	+	Chronic HB with low Replication
-	-	IgM	+ or -	-	Acute HB
-	+	IgG	-	+ or -	Recover

Antigens

1. Surface Antigen (HbsAg).

   It is detectable from the “first month” of infection in the incubation period.

2. e- Antigen (HbeAg).

   It is a water soluble form, from the protein of core antigen It is detectable from the “first half of the second month” of infection (incubation period).

   It is indicate about the virus replicating and associated with present of HBV-DNA .

   Its present beyond 6 month suggests chronic hepatitis

Antigens

3. HBV-DNA Genome.

It is detectable in serum from the “first month until chronic stage”. A positive test indicates that the virus is multiplying in a person’s body and that the person is highly contagious. The test is often used to monitor the effectiveness of antiviral therapy in people with chronic HBV infection (it is detectable by PCR).

4. Core Antigen (HbcAg).

It is not found in the blood.

Anti-Bodies Response

1. Suface anti-body or anti-HBS (anti-HbsAg).

   It is detectable from the “sex month” after disappearance HbsAg and remain for life.

   It is response to:-

   A. Successful Vaccination.

   B. Pervious HB infection (not found in carriers).

   C. Some times may be detectable in chronic infection simultaneously both Anti-HBS and HbsAg.

2. "e"- Antibody or Anti-Hbe (Anti-HbeAg).

   It is detectable from “fourth month” and remain for life.

   It is indicate that the virus "B" stops replicating in the liver.

Anti-Bodies Response

3. Anti-HBC or core antibody (Anti-HbcAg).

It is detectable from the "first month " of infection after the appearance of HbsAg and remain through the diseases stage and after recovery.

It is response to:-

A. Carrier at birth.

B. Has been infected by the virus "B".

C. Currently infected by the virus "B".

Its Persists even after anti-HBS not detectable, and its persist means that there is no need for vaccination.

Anti-Bodies Response

4. IgM Anti- HBC.

It is a routine test used to confirm the diagnosis of acute HBV infection.

IgM Anti-HBC is important when patients with acute liver failure due to HBV and may be not present HbsAg , So in this case the IgM anti- HBC may be the only one marker can detect about the HBV infection.

Levels of IgM anti -HBC may rise in patients treated with anti viral such as INTREFERON or in those undergoing spontaneous seroconversion.

HEPATITIS “B” VACCINE

Nature of vaccine are:-

1. Recombinant HB vaccine (Ingerix)

It is produced by common ” Backer's Yeast” in with plasma containing the gene for HbsAg protein

or it is a vaccine which produced by DNA technology contain the gene for HbsAg.

It is a very effective and protective.

Dose:- 0, 1, and 3-6 months

Booster Dose:- On 5th Year

HEPATITIS “B” VACCINE

2. Plasma derived HB Vaccine

   It is an inactivated HbsAg protein purified from “human plasma”.

   Duration of action:-

   3-5 years, So there is need for BOSTER dose after five years.

3. Hepatitis B immune globulin (HBIG) – It is recommended for persons who have had exposed to hepatitis “B”.

Specific Treatment

1. Interferon
   - Interferon alpha, beta , gamma
   - Peglytaed Interferon alfa 2a & 2b (Peg IFN).
2. Nucleoside and nucleotide analogues
   - Ribavirin (virazol 200mg)
   - Epivir (lamivudine 100mg, filmatalen)
   - Entecavir and Tenofovir- have high potency and tolerability
3. Surgery intervention
   - Resection
   - Transplantation

Mechanism of action of interferon

1. Anti - Proliferate effect (anti proliferation, anti- multiplication of cells)
2. Anti-viral effect (interferes or inhibits viral DNA polymerize)
3. Immune-modulator effect (stimulation)
4. More effective in “CAH” patients with high aminotransferase.
5. Unsuccessful for those who are acquired infection from the birth (HbsAg carriers at birth).
6. Unsuccessful for those who are HIV positive.
7. Unsuccessful for those who are Immune- deficient.

Interferon is administered by subcutaneous injection three time or once (peglytaed) weekly in combination with Ribavirin

Indication to interferon therapy

1. Elevated HbsAg , HbeAg , DNA virus by PCR
2. Elevated liver ALT (GPT) enzyme
3. Moderate chronic hepatitis
4. liver fibrosis (well compensated)

Contraindication : Decompensated cirrhosis

Side - Effect

1. Flu-like syndrome (fever, fatigue, nausea, cough and sinusitis)
2. Lethargy
3. Insomnia
4. Diarrhea
5. Depressive symptom
6. Thyroid disorders
7. Hematological disorders such as neutropenia, thrombocytopenia and hemolytic anemia
8. Impotence
9. Rheumatoid arthritis
10. Skin disorders (psoriasis ,vitiligo, lichen planus, itching and skin rash)
11. Cardiovascular disorders
12. Psychosis and irritability.
13. Seizures
14. Peripheral neuropathy

Contraindication

To Interferon

- Severe cardiac disease
- Renal failure
- Decompensate liver disease
- Epilepsy
- Severe neurological impairment
- Uncontrolled thyroid disease
- Severe psychiatric disease particularly Depression

Contraindication

To Ribavirin

- severe cardiac disease
- Renal failure
- Decompensate liver disease
- Pregnancy
- Breast feeding
- Haemglobinopathies

Problems of treatment by interferon

1. Remain expensive 2. Have toxic side effect
2. Low patient responding 3. Remain the risk of relapse
4. Rarely total elimination of the virus 5.Thrombocytopenia

Immunization of contacts:

HBIG 5ml I\M for adults or 0.06mg\kg within 24 hours after exposed to blood or any contact, second dose month after.

Signs of Responding

1. Low HBV-DNA during the second or third month of therapy.
2. High baseline ALT during the second or third month of therapy.
3. Fall the level of HbeAg while the level of Anti-Hbe Rise
4. Rise the level of IgM Anti-HBc.
5. At the end of the course of treatment

   HBV -DNA should be Negative and liver enzymes are nearing Normal.

Note: We should do blood analysis every month to insure blood cells

End-Point

Loss of HbeAg, negative test of DNA and development of Anti-HBe these events mark the end of viral replication , even the HbsAg remain continue it will cease in time.

Also regression of fibrosis and normalization the risk of cirrhosis, hepatocillular carcinoma (HCC) and LFT.

Viral Hepatitis “C”

Definition

Hepatitis “C” virus infection is a common worldwide problem that may progress to chronic hepatitis in 75%, cirrhosis in 25% and hepatocillular carcinoma in 1 - 4% with the subsequent development of complication such as ascites, encephalopathy and variceal bleeding.

Most of Patients diagnosed years later (5-30 years) with complication of Chronic hepatitis and cirrhosis

Epidemiology

1. Hepatitis “C” virus infection is a common worldwide problem affecting 170 million population or about 3% (carriers).

2. Hepatitis “C” virus infection is endemic in most regions of the world with the prevalence of HCV in Africa 5,3%, in Americas 1,7%, in Eastern- Mediterranean 4.6%, in Europe 1.03 in South-East Asia 2.15%, in Pacific 3.9%.

Epidemiology

3. **The prevalence of HCV in Yemen about:-**

   2- 6% while approximately 5% in Saudi Arabia and 1.8 % in the USA those also who are at risk of developing liver cirrhosis and or liver cancer.

4. **Hepatitis "C" virus infection primarily transmitted by blood and blood product 90%, I\V drug abuse, vertical transmission and sexual contact (low risk). Transmission via breast feeding has not been documented.**

Virology

1. hepatitis “C” virus infection is “ RNA virus” and a member of the flaviviride family
2. The RNA viruses have high mutation rates
3. The RNA viruses have different Genotypes, There are 6 known Genotypes as follow:-
    a. Genotype 1 is about 75 % -common in America
    b. Genotype 2 is about 10% - common in Europe
    c. Genotype 3 is about 10% -common in Europe
    d. Genotype 4 – common in Central Africa and Middle East
    e. Genotype 5 - common in South Africa
    f. Genotype 6 – common in Hong Kong and Vietnam
4. There is a little difference in mode of transmission among the different genotypes, but there is a large difference in response to interferon medication.

Diagnostic Testing

1. Anti-body to multiple HCV antigens (HCV enzyme immunoassay) within 6 weeks
2. Hepatitis C virus RNA by polymerize chain reaction (PCR) within 2 weeks or transcription-mediated amplification(TMA) to confirm the presence of “viremia”. The level of HCV RNA in blood helps in predicting the response and change in the level of HCV RNA during treatment.
3. Liver biopsy – To detect the severity of the disease especially fibrosis , cirrhosis and hepatocillular carcinoma)
4. Genotypes testing.

Clinical picture & Natural History

1. The majority of hepatitis “C” virus cases are asymptomatic.
2. Nonspecific symptoms such as fatigue, vague, right upper quadrant discomfort and pruritus may be noted.
3. Acute hepatitis “C” : Uncommon.
4. Not all patients progress to cirrhosis in fact approximately 25% progress to fibrosis and cirrhosis and 1-4% of them develop hepatocellular carcinoma (Hepatoma).
5. The other 75% have varying degree of chronic hepatic inflammation.

Clinical picture & Natural History

5. The average duration of hepatitis “C” infection before the onset of cirrhosis 5-50 years.

6. Factors that contribute to the development of cirrhosis are unclear but thought following:-
    a. Genetic factor
    b. Alcohol consumption
    c. Obesity

Clinical picture & Natural History

7. Neither aminotransferase elevation nor viral load correlate the inflammatory activity or the present of fibrosis in fact, But the only one way to asses disease severity is the LIVER BIOPSY.

8. The surgical therapy(resection or liver Transplantation) of hepatocillular carcinoma offers the only hope for cure.

9. The others screening like using ultrasound , Magnetic resonance imaging (MRI) with gadolinium, triphasic computed tomography scan is Necessary for diagnostic.

Specific Medical Therapy

Indication **to "interferon"** therapy

1. Elevated ALT (GPT)
2. Moderate CH,
3. RNA by PCR
4. compensated fibrosis .

Types of Interferon

1. Peglytaed interferon alpha 2a 180 mg per week, subcutaneous injection.
2. Peglytaed interferon alpha 2b 1,5mg/kg per week, subcutaneous injection.

HARVONI COMBINATION

Harvoni combination (sovaldi +lidipasvir) tab. 1x1 per day for 8,12, 24 weeks – Its effective about 99 % especially in treating genotype -1 HCV.

Ribavirin

It is administered orally in divided dose by weight for Genotype I and 4 (1000mg/day if <75 kg and 1200mg/day if > 75kg) whereas for Genotype 2 and 3 only 800mg /day regardless of weight, Ribavirin must given in combination with interferon.

Epivir (lamivudine)

Orally 100mg per/day for at least one year.

Course of therapy

- For Genotype 1 and 4 must receive a 48 week course, But for Genotype 2 and 3 must receive only a 24 week course of medical therapy.

Sustained Response Rate

1. 42-46 % for Genotype 1 and 4
2. 78-82 % for Genotype 3 and 2
3. 54-56 % average

Epivir (lamivudine)

The Decision of initial treatment

Must be taking into account an individual base as follow:-

1. Patient motivation
2. Severity of disease
3. Likelihood of attaining a response
4. Contraindication to therapy

Treatment Updating: Daklinza, Harvoni, Sofosbuvir (Sovaldi), Simeprevir (Olysio), and Viekira Pak

Indication for initial treatment

1. No Cirrhosis, Raise ALT, RNA and moderate CH
2. Patient with initial Fibrosis without Decompensated by US, MRI and liver biopsy.
3. Patient with any Genotypes of HC viruses
4. Patient with flu-like symptoms
5. Patient with extra hepatic manifestation

Note:- **It is unclear yet if therapy is necessary for Asymptomatic patients with Genotype 1 and no hepatic fibroses.**

Adverse Effects of specific therapy

Side effect of Interferon

- Fatigue, headache, fever and chills, nausea, weight loss, alopecia, irritability, depression, injection site reaction, autoimmune disease exacerbation (eczema, psoriasis, thyroid disease, hypothyroid and hyperthyroid, others), neutropenia and thrombocytopenia
- **Contraindication** : That mention before

Ribavirin

Cough, shortness of breath, rash, nausea, weight loss, hemolytic anemia and teratogensis.

Early Stopping Treatment:

Patients who will not response in therapy within 12 weeks of therapy by declining quantitatively HCV RNA titers more than 2 logs, medical therapy should be discontinued in all genotypes of hepatitis.

If HCV RNA is detectable at week 24 for genotype- 1, Medical therapy should be discontinued

Contraindication : That mention before

Autoimmune Hepatitis

AH is a chronic disorder characterized by continuing hepatocellular "Necrosis" with fibrosis leading to cirrhosis and hepatic failure as a result of autoimmune process against liver antigen

Diagnosis:- blood antibodies of ASMA, ANA, Antiactin, Anti-LKM and Soluble liver antigen

Clinical Picture:- may present with signs of Chronic liver disease, Hepatosplenomegaly, Cushing face with Acne and Hirsutism, Jaundice, Spider Nevi and Ascites

Cirrhosis

Cirrhosis is an irreversible chronic parenchymal disease of liver resulting from the “Necrosis” of liver cells followed by fibrosis and nodule format

Etiology:- 1) Chronic hepatitis “B” , “C”, “D” 2) Alcohol

3) Hemochromatosis 4) Wilson’s disease 5) Alpha – 1 antitrypsin deficiency 6) Biliary obstruction primary and secondary 7) Autoimmune Hepatitis 8) Drugs

Clinical Feature:- 1) Non- specific As Chronic hepatitis

2) Specific As a) Portal Hypertension b) Hepatic Insufficiency c) Ascites d) Palmar Erythema e) Spider nevi f) Impotence

g) Gynecomastia h) Breast atrophy h) Variceal Bleeding

Hepatitis “D” and “G”

- Hepatitis “D”
- It is RNA virus requires hepatitis “B” for replication.
- Similar to hepatitis “B” in:- mode of infection, diagnosis, clinical signs, prophylactic, treatment and complication
- Simultaneous with HBV or as a super infection in chronic carriers of hepatitis “B” virus
- Hepatitis “G”
- Transmitted through skin , mostly affected persons with I\|V drug abuse and hemodialysis patients, causes mild hepatitis, leads to chronic hepatitis

  Diagnostic tests are not available yet.

Additional Reading

- 1. Sheila Sherlock
  Disease of the liver and biliary system
  Blackwell Oxford UK.ISBN 0-632-02443-7.

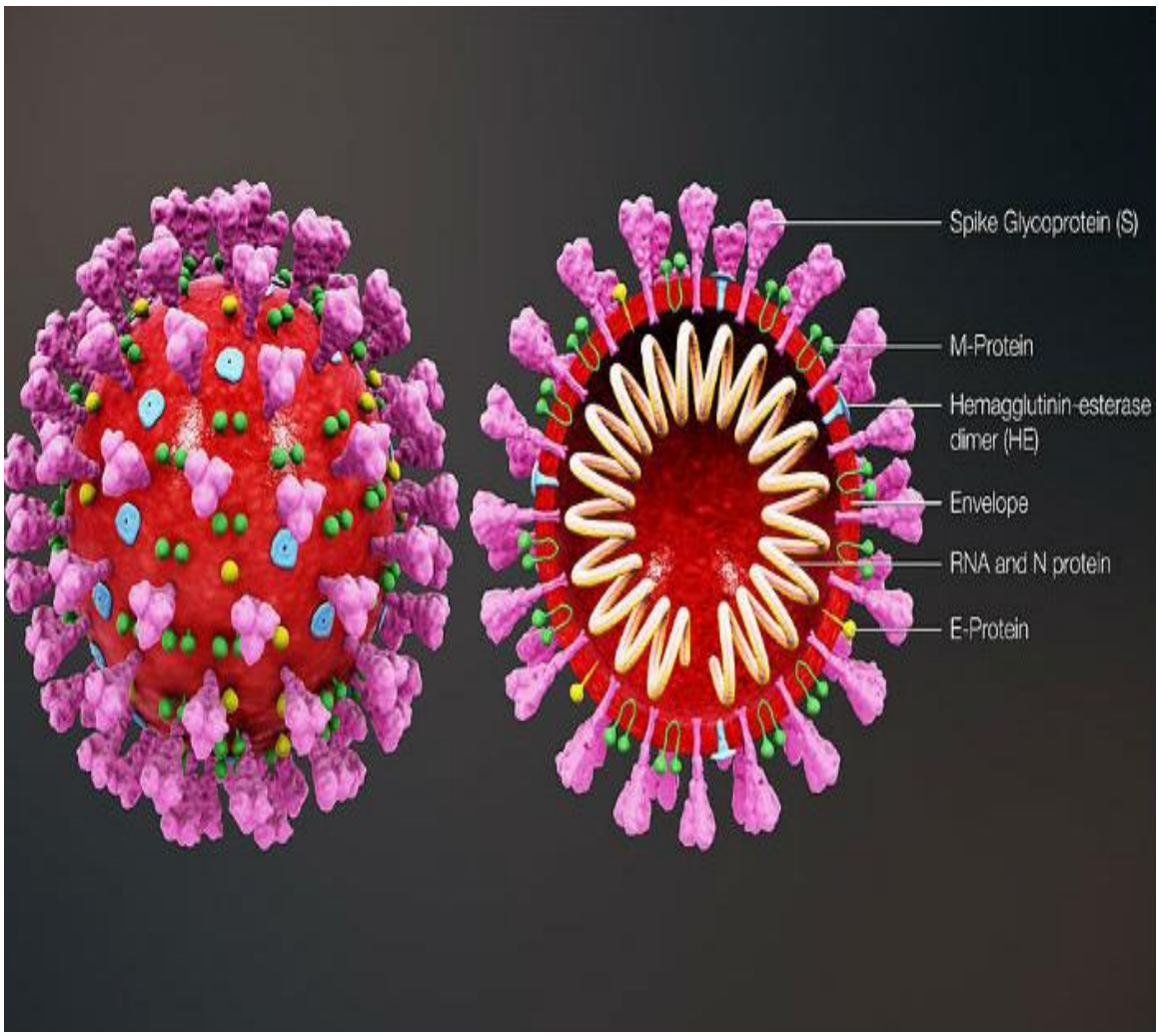
2. Mohamed Imam Danish - 1917. Pakistan
Medical Diagnosis and Management

3.Christopher J.T. & Heather M.S.
Clinician's Guide To Viral hepatitis
London .2002. ISBN 0340763043

4, Steven L.Flamm
Chronic Hepatitis C Virus Infection
JAMA September 2003p 42-46.

5. Ossama A. Shobokshi
Hepatitis C virus genotype 4
Saudi medical Journal.V.24.Supllement .2.

Corona virus structure



CORONA Virus – Covid19

Dr. Ahmed M. Al - Haddad

Definition

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CORONA Covid 19 is an acute novel viral infection of respiratory system leading to an atypical pneumonia.

- Corona covid 19 causes blood-clotting and severe abnormalities , which can damage lung, heart and other major organs throughout the body

Causative agent

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Corona - Covid 19 – virus from corona family, RNA (previously unidentified)

It is a big virus, size 400- 500 nm (nanometer), a thousand of micrometers (polio virus 25-50nm, viral hepatitis A and B 25, 28 nm respectively)

Now a day Covid 19 have a lot **of variants** such as:

1) Alfa - Announced about it in England - September 2020
2) Beta – Announced about it in south Africa - May 2020
3) Gama – Announced about it in Braze lea-November 2020
4) Delta - Announced about it India – October 2020
5) Omicron – Announced about it in south Africa and many countries – November 2021
6) ba2, ba1, ba1.1,ba3 etc… - Announced about it in England and other countries - January 2022 (all these variants divided from omicron variant)

Reservoir of infection

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Human case and carriers

Wild animals (Corona-Covid 19 transferred from animal to human)

Source of infection

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Respiratory secretions

Epidemiology

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Age: All ages, but more in old ages

Sex: Both sexes

Susceptibility: All groups

Post infection: Unknown yet

Place: Worldwide

1) A worldwide alert on corona covid-19 was issued Chinis authorities late last year (December 2019)

2) Corona - Covid - 19 recognized in the late of year of 2019 in Wuhan, China and since that till now (1 June 2021) has made its way into all courtiers outside main land china and has so far infected many people

3) Corona-Covid -19 outbreak began late of yea**r 2019** (December - 2019) and considered as a new emerging communicable disease of the 20st century resulted pandemic by the beginning of 2020. when**" WHO" announced it's officially as a Pandemic infection on 11 March 2020**

4) Globally it's was recorded till today (1 June 2021) Corona covid 19, 182.5 million cases, out of them 3.9 million death (Case fatality rate 4.6 %), tell now.

5) In Yemen first case recorded on Friday 10 .4. 2020 in Hadramout, till now 1 of June 2021 recorded by Yemen national committee of corona protection 6923 cases, 1361 deaths (case fatality rate 19.6 %)

6) The disease spread from Asia to American continent, Europe and all countries in the world **through International Travels**

7) History of travel to an area with recent local transmission of Corona covid-19.

8) Route of transmission are mouth, nose and eyes

9) The distance between cases or carriers and healthy person Must be not less than 2 meters to protect the infection

Registration of the 10th Priorities countries Corona covid 19 by Death, Cases and Case Fatality Rare (Jon Hopkins University Report in the beginning of March 2021) .

Country	Deaths	Cases	CFR
U.S.A.	524. 987	28.991.537	1.8
Brazil	265.411	11.019.344	2.4
Mexico	190.357	2.125.866	8.9
India	157.756	11.210.799	1.4
England	124.736	4.231.166	2,9
Italy	99.785	3.067.486	3.2
France	88.727	3.964.078	2.2
Russia	87.614	4.274.263	2.0

Germany	71.956	2.508.655	2.8
Spain	71.138	3.149.012	2.2
Total	2.592.768	116.817.145	2.2

Period of communicability

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Short limited hours or days

Corona - Covid 19 survive outside of human body:

It stays about 2-3 hours on the droplets, 8 hour on the air, but on the poop, materials or equipment it stay about 9 days

Corona - Covid19 may die under 30c of hot and under 60c of cold.

Incubation Period

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2 - 14 days

Mode of transmission

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1) By Droplets transmission and airborne transmission
2) By Close contact with patients or carriers (person to person)
3) By Touching any objects, materials and equipments contaminated by Corona - Covid 19, Then touching nose, mouth and eyes

Predisposing Factors (People at risk more)

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1) Persons in old ages
2) Patients with chronic diseases
3) Immunodeficiency person
4) Malnutrition people
5) Smoking, alcohol drinking, khat chewing, Shisha and Shama'a
6) Big meeting, meeting should be not more than 50 persons
7) Medical staff
8) Medical health care

Firstly sex signs of corona covid 19 (Mild)
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1) Itchy and Sore throat
2) Dry throat
3) Dry cough
4) Fever mostly high
5) Shortness of breath
6) Loss of taste and smell

Clinical Picture of mild cases
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1) High Fever
2) Cough usually dry
3) Chest pain
4) Water eyes or runny eyes
5) Fatigue
6) Headaches
7) Sore throat
8) Difficulty in breathing or
9) Shortness of breath
10) Dry mouth and throat
11) Sometimes gastrointestinal symptoms could be the First signs of Covid 19 infection (vomiting, nausea and diarrhea) before patients get the fever.
12) Runny nose
13) Temporary loss of taste and smell
14) Inflammatory of blood vessels in the hands and feet's (Nicknamed covid 19 toes)

Prognosis
----------.

90 - 95 %	Unapparent infection (Symptomless)
5 - 8 %	Mild illness - need health care (Home)
2 -5 %	Severe illness - need intensive care (Hospital)
0.5 -3 %	Death - Case fatality rate

Complication

----------.

Pneumonia, Respiratory failure, renal failure and may be death

Diagnosis

----------.

1) Careful epidemiological picture e.g. travel history or contact with a person diagnosed with corona patient or carrier
2) Careful clinical picture
3) Viral culture
4) Existing ELIZA test
5) Existing PCR tests
6) Chest X-Ray (CXR)

Management

----------.

A) Mild cases - treatment Home

----------.

1) Home isolation with suitable meal and drink
2) Panadol 1000 mg 1x2 per day
3) Azitromycine 500 mg 1x1 for 5 days
4) Vitamin ”C” 1000mg 1x2 per day
5) Zinc 50 mg 1x1 per day
6) Vitamin “D” 50000 one tab per week
7) Aspirin 75-100 mg 1x2 daily
8) Strepslis 1x3 per day

B) Severe cases - Hospitalization

----------.

1) Severe Cases: Supportive treatment especially rehydration and oxygen therapy in specific Quarantine
2) Treat symptoms and complications in some countries such as Hydroxy- Chloroquine +Azitromycine (Azitone) + Vit.C. with specific therapy in specific hospital
3) A case should be excluded if an alternative diagnosis can fully explain the illness

4) Specific therapy anti viral drugs such as Remdesivir and Backslowvid etc…

Prevention and Control

----------.

1) Vaccinations which started just by December 2020 and now it's worldwide such as Modern'a Pfizer, AstraZeneca, and Sputnik etc…..
2) Frequently Hand Washing with soap, wearing gloves, and surgical mask
3) Throat and sore cleaning with hot water and salt or "Betadine"
4) Disinfected any poop, subjects, materials, equipments and clothes in the contaminated area (by Ethanol, hydrochloride sodium, peroxide hydrogen etc..)
5) Social distancing and avoiding overcrowding places as buses, lecture hall, school classes, celebrating houses, population market and avoid close contact with people who are at risk, poor ventilated rooms, kissing others etc...
6) Eating vegetables and fruits retches vitamin "C"
7) The patient should be moved to a single room with the door closed
8) People with chronic diseases especially old ages should be stay home in the outbreak of Corona Covid -19
9) Medical Staff should wear a surgical mask
9) Patient should wear a surgical mask until they are admitted to the isolation room
10) Health workers staff should immediately inform the Infection to the Control department that a patient is under investigation for Corona
11) Eye protection is strongly recommended (e.g. goggles)
12) Patient should not be left sitting in the waiting room area
13) A suspect case should transported immediately to the hospital and the hospital should be notified of the patient 's pending arrival
14) Local public health authority should notified immediately of the case

15) Any one who accompanied the suspect case to the outpatient setting should be given written information on infection prevention and control measures related to Corona
16) Isolation Probable and suspected cases in the hospitals or in health centers
17) Quarantine in the Airport and all ports in the country
18) Disinfection procedure for materials and equipment
19) Area should be under surveillance
20) Avoid smoking, alcohol, chat chewing, Shisha and Shama'a
21) Immediately international notification especially WHO

Epidemiology and control of Leishmaniasis

Leishmaniasis

Are a group of protozoal diseases caused by parasites of the genus leishmania, and transmitted to man by the bite of female phlebotomine sandfly.

Forms of leishmaniasis

There are 3 main forms of leishmaniasis

I. **Visceral leishmaniasis** (VL), also known as kala-azar is fatal if left untreated in over 95% of cases. It is characterized by irregular attacks of fever, weight loss, enlargement of the spleen and liver, and anemia.

Most cases occur in:

- South America,
- South Africa,
- The Mediterranean countries,
- India etc.

Forms of leishmaniases – cont.

II. **Cutaneous leishmaniasis** (CL) is the most common form of leishmaniasis and causes skin lesions, mainly ulcers, on exposed parts of the body, leaving life-long scars and serious disability or stigma.

About 95% of CL cases occur in:

- The Americas,
- The mediterranean basin,
- The middle east and central Asia.

Forms of leishmaniases – cont.

III. **Mucocutaneous leishmaniasis** leads to partial or total destruction of mucous membranes of the nose, mouth and throat.

Over 90% of mucocutaneous leishmaniasis cases occur :

- Bolivia,
- Brazil,
- Ethiopia,
- Peru.

Epidemiological situation

- The overall prevalence is 13 million cases and estimated population at risk is about 350 million.
- An estimated 700 000 to 1 million new cases and some 26 000 to 65 000 deaths occur annually.
- Worldwide there is an increasing number of cases and more widespread geographical distribution, the disease being reported from previously non-endemic areas.

Epidemiological situation – cont.

- Leishmaniasis is found in people in focal areas of approximately 90 countries in the tropics, subtropics, and southern Europe.
- Economic and demographic circumstances that contribute to increase prevalence include:
  - ✓ New agro - industrial projects,
  - ✓ Large – scale migration of populations,
  - ✓ Unplanned urbanization,
  - ✓ Man – made environmental changes.

Epidemiological situation – cont.

- Co –infection of visceral leishmaniasis and AIDS is emerging due to spread of the AIDS pandemic particularly in Southern Europe, where 25 to 70% of adult visceral leishmaniasis cases are related to HIV infection, and 1.5 to 9% of AIDS patient suffer from newly acquired or reactivated visceral leishmaniasis.

Epidemiological situation – cont.

- The disease affects some of the poorest people on earth, and is associated with:
  - ✓ Malnutrition,
  - ✓ Population displacement,
  - ✓ Poor housing,
  - ✓ A weak immune system,
  - ✓ Lack of financial resources.

Epidemiological situation – cont.

- Leishmaniasis usually is more common in rural than in urban areas, but it is found in the peripheries of some cities.
- Climate and other environmental changes have the potential to expand the geographic range of the sand fly vectors and the areas in the world where leishmaniasis is found.

Causative agent

- Leishmaniasis is caused by the protozoan leishmania parasites.
- At least 19 different leishmania parasites have been associated with human infection.
- The majority of these offer no cross immunity of one against the other.
- Leishmaia donovani, L. infantum and L. chagasi are the causative agents of visceral leishmaniasis (kala-azar),
- L. tropica, L. major, L. aethiopica are the causative agents of cutaneous leishmaniasis (Old world, orient sore),

Causative agent – cont.

- L. braziliensis and L. Mexicana are the causative agents of muco-cutaneoous leishmaniasis (New world).
- The leishmania are intercellular parasites.
- Visceral forms may produce cutaneous lesion, and cutaneous forms may produce visceral forms.
- The life cycle is completed in two different hosts:
  - A vertebrate,
  - An insect.

Reservoirs

Some 70 animal species, including humans, have been found as natural reservoir hosts of leishmania parasites:

- Man,
- Domestic dogs,
- Foxes,
- Wild rodents,
- Other mammals.

The majority of the leishmamiasis are zoonoses.

Environmental factors

- Altitude: It does not occur in altitude over 600 meters above the sea level,
- Season,
- Rural areas,
- Vectors,
- Development projects,
- Leishmaniasis is linked to environmental changes such as deforestation, building of dams, irrigation schemes, and urbanization.

Mode of transmission

- Leishmania parasites are transmitted by the bite of infected female phlebotomine sandflies.
- The sand fly vectors generally are the most active during dusk, evening, and night-time hours (from dusk to dawn).
- Over 90 sandflies species are known to transmit leishmania parasites.

Host factors

- Age

Leishmaniasis can occur in all age groups including infants.

- Sex

Males are affected twice as often as females.

- Population movement

Movement of population (migrants, laborers, tourists) between endemic and non-endemic areas can result in the spread of infection.

Host factors – cont.

- The disease strongly associated with occupation.

:People who work in

  - Various farming practices,
  - Forestry,
  - Mining,
  - Fishing have a great risk of being bitten by sandflies.

- Immunity

Risk factors

- **Socioeconomic conditions**
  - ✓ Poverty increases the risk for leishmaniasis.
  - ✓ Poor housing and domestic sanitary conditions may increase sandflies breeding and resting sites, as well as their access to humans.
  - ✓ Sandflies are attracted to crowded housing as these provide a good source of blood-meals.
  - ✓ Human behavior, such as sleeping outside or on the ground, may increase risk.

Risk factors – cont.

- **Malnutrition**

Diets lacking protein-energy, iron, vitamin A and zinc increase the risk that an infection will progress to a full-blown disease.

- **Environmental changes**

The incidence of leishmaniasis can be affected by changes in urbanization, and the human invasion into forested areas.

Risk factors – cont.

- **Population mobility**

Epidemics of both cutaneous and visceral leishmaniasis are often associated with migration and the movement of non-immune people into areas with existing transmission cycles.

Occupational exposure as well as widespread deforestation remain important factors.

Risk factors – cont.

- **Climate change**

Leishmaniasis is climate-sensitive as it affects the epidemiology in a number of ways:

- Changes in temperature, rainfall and humidity can have strong effects on vectors and reservoir hosts by altering their distribution and influencing their survival and population sizes,
- Drought, famine and flood can lead to massive displacement and migration of people to areas with transmission of leishmania, and poor nutrition could compromise their immunity.

Incubation period

- At least a week, to many months for cutaneous and mucocutaneous leishmaniasis.
- 2-4 months, range is 10 days to 2 years for visceral leishmaniasis.

Period of communicability

- Cutaneous and mucocutaneous leishmaniasis – As long as parasites remain in lesion, in untreated cases, usually 5 months to 2 years.
- Visceral leishmaniasis – As long as parasites persist in the circulating blood or skin of the mammalian reservoir host. If man is the reservoir host, infectivity for sandflies may persist even after clinical recovery.
- Transmission from person to person, and by blood transfusion and sexual contact have been reported.

Susceptibility and resistance

- Cutaneous and mucocutaneous leishmaniasis – susceptibility is probably general.
- Some immunity is present after lesion heal, it is not clear how protective this might be against other species.

Prevention and control

Preventive measures:

- Educate the public concerning mode of transmission and methods of controlling sandflies and mobilization of the community with effective behavioral change interventions.

Preventive and control measures – cont.

- Control measures vary from area to area depending on the habits of the mammalian host and the vector. Where the habits are known, applicable control measures may be carried out. These include:
  - Systematic early case detection and rapid, effective treatment.
  - Periodic application of insecticides with residual action.
  - Elimination rubbish heaps and other breeding places for the old world vector.

Prevention and control – cont.

- Elimination of domestic canine reservoir.
- Destroy animals (and their burrows) implicated as principal reservoirs in local area.
- In the new world, avoid sandfly-infested and thickly forested areas, particularly after sundown, use insect repellents and protective clothing if exposure to sandflies is unavoidable.
- Effective disease surveillance.
- Apply appropriate environmental managements and forest clearance as possible.

Epidemiology and control of Tetanus (lockjaw)

Dr. Abdulla A. Muharram

Tetanus is an acute infectious disease of the nervous system, induced by the exotoxin of clostridium tetany, often fatal disease marked by tonic muscular spasms and hyperreflexia, resulting in trismus (lockjaw), generalized muscle spasm, opisthotonos, glottal spasm, seizures, respiratory . spasms

Background

- **Tetanus is a global health problem, as *C. tetani* spores are ubiquitous in soil and animal feces.**
- **The disease may occur at any age and case-fatality rates are high even where intensive care is available.**
- **There are about one million cases of tetanus reported worldwide annually.**
- **In the absence of medical intervention, the case-fatality rate approaches 100%.**

Background – cont.

- **The majority of reported tetanus cases are birth-associated, occurring in low income countries among insufficiently vaccinated mothers and their newborn infants, following unhygienic deliveries and abortions, and poor postnatal hygiene and cord care practices.**
- **Deliveries carried out by persons with un-cleansed hands or on a contaminated surface are also risk factors.**

Background – cont.

- **The disease remains an important public health problem in many parts of the world where immunization programmes are suboptimal, particularly in the least developed districts of low income countries.**
- **The elderly and diabetes are increased risk for disease.**

Background – cont.

- **Neonatal tetanus is a problem in underdeveloped communities where deliveries are usually carried by traditional birth attendants, and asepsis is not followed.**

- **The spores can also be found on skin surfaces and in contaminated heroin.**

Causative agent: *Clostridium tetani*

- **An aerobic gram-positive, spore-forming bacteria.**
- **Spores found in soil, animal feces, may persist for months to years.**
- **Vegetative state produces two exotoxins:**
  - ✓ **Tetanolysin,**
  - ✓ **Tetanospasmin.**
    - **Tetanospasmin-Neurotoxin responsible for the clinical manifestations of tetanus.**
- **Tetanospasmin estimated human lethal dose = 2.5 ng/kg.**

Reservoir of infection

- **The natural habitat of the organism is soil and dust.**

- **The bacilli are found in the intestine of herbivorous animals (mainly) and humans (occasionally) and excreted in their feces.**

Neonatal Tetanus

Is a form of generalized tetanus that occurs in newborn infants.

It occurs in infants who have not acquired passive immunity because the mother has never been immunized.

- **It is a killer disease, second only to measles among the target diseases of the EPI.**

- **The case - fatality rate can be as high as 80 – 90%.**

Neonatal Tetanus – cont.

- **WHO estimates that in 2015, approximately 34000 neonates died from neonatal tetanus.**

- **It tends to occur in areas with poor access to health care, hence it often remains hidden with the community.**

Modes of transmission

Contamination of wounds with tetanus spores by different methods :

1 - Injury in polluted places,

2 - Neonatal infection through :

- **Using improperly sterilized catgut (catgut is prepared from sheep gut).**

Modes of transmission – cont.

- **Contamination of wound with tetanus spores – containing dust, from unclean hospital environment and surrounding area.**
- **Reactivation of dormant tetanus spores in contaminated old surgical intervention.**

3 – Surgical tetanus,

4 – Puerperal infection.

Environmental and social factors

- **Tetanus is a positive environmental hazard.**
- **Its occurrence depends upon man's physical and ecological surroundings – the soil, agriculture, animal husbandry – and not on the presence or absence of the infection in the population.**

The environmental factors are compounded by social factors such as unhygienic customs and habits (e.g., application of the dust or animal dung to wounds).

Types of tetanus

- **Traumatic,**
- **Puerperal,**
- **Otogenic,**
- **Idiopathic,**
- **Neonatal.**

Susceptibility and resistance:

Old and young male and female are all susceptible.

Period of Communicability:

It is not communicable from man to man.

Incubation period:

Varies from 4 days to 21 days or more. Average is one to three weeks. Short incubation period is more serious and fatal.

Tetanus clinical features

Three clinical forms:

- **Local (not common),**
- **Cephalic (rare),**
- **Generalized (most common).**

- ✓ **Generalized tetanus: descending symptoms of trismus (lockjaw), difficulty swallowing, muscle rigidity, and spasms,**
- ✓ **Spasms continue for 3-4 weeks,**
- ✓ **Complete recovery may take months.**

Trismus and Sardonic Smile

Opisthotonos

Tetanus Complications

- **Laryngospasm.**
- **Fractures.**
- **Hypertension.**
- **Nosocomial infections.**
- **Pulmonary embolism.**
- **Aspiration pneumonia.**
- **Death.**

Diagnosis

The diagnosis of tetanus made on:

- **Epidemiological data.**
- **Clinical data.**

Prevention:

- General measures.
- Specific prevention.
- Care of wound.
- Prevention of different forms of tetanus.

General measures :

1 -Community development and clean environment, urban and rural.

2 -Segregation of stables and animal sheds from residential areas.

3 -Replacing animal carts with mechanical vehicles.

Specific Prevention :

1 -Immunization.

a -Active Immunization.

b-Sero-prophylaxis.

2 -Chemoprophylaxis.

Active immunization By :

1. **Tetanus toxoid,**
2. **D P T,**
3. **Pentavalent,**
4. **D T.**

Indications :

1. **Military forces,**
2. **Occupational exposure,**
3. **Pregnant women.**

Tetanus toxoid immunization schedule for women of childbearing age and pregnant women who haven't received DPT/Penta vaccine

Dose of TT or Td (according to card or History)	When to give	Expected duration of protection
1	At 1st contact or as early as possible in pregnancy	None
2	At least 4 weeks after 1st dose	years 1-3
3	At least 6 months after 2nd dose or subsequent pregnancy	years 5
4	At least 1 year after 3rd dose or subsequent pregnancy	Years 10
5	At least 1 year after 4th dose or subsequent pregnancy	For all childbearing age years and even longer

Td/TT immunization schedule for women who were immunized during infancy, childhood or adolescence

Age at last vaccination	Previous vaccination (based on written Record)	Recommended Immunization	
		At present Contact/pregnancy	Later (at least at 1 year interval)
Infancy	DPT/Penta doses 3	doses of Td/TT 2 (min 4 weeks interval)	dose of Td/TT 1
Childhood	DPT/Penta doses 4	dose of Td/TT 1	dose of Td/TT 1
School age	DPT + 1 DT/Td 3	dose of Td/TT 1	dose of Td/TT 1
School age	4 DPT+ 1 DT/Td	dose of Td/TT 1	None
Adolescenc	4 DPT + 1 DT at 4-6 yrs	None	None

Sero-prophylaxis:

1)Tetanus antitoxin (animal),

2)Human antitoxic immunoglobulin.

Chemoprophylaxis:

Penicillin can be given in place of sero-prophylaxis to prevent local activity of tetanus bacilli in wounds.

:Care of wounds

Early management of wounds soon after injury (aseptic cleaning, and removing any foreign matter and necrotic tissue, which would otherwise favor activity of tetanus bacilli).

Prevention of other forms of tetanus

A – <u>Neonatal tetanus :</u>

- **Aseptic cutting the cord,**
- **Care of umbilical stump, using sterile dressing,**
- **Training of midwives and traditional birth attendants,**
- **Active immunization of mothers.**

Prevention of other forms of tetanus – cont.

B – <u>Surgical infection :</u>

- **Clean hospital environment and surrounding area,**
- **Asepsis and sterilization throughout all procedures,**
- **Precise manufacture of sterile catgut,**
- **Care and protection of wounds.**

Prevention of other forms of tetanus – cont.

C – <u>Puerperal infection :</u>

- **Asepsis and sterilization in labour or abortion,**
- **Active immunization of pregnant women in high – risk areas,**
- **Chemoprophylaxis after labour or abortion.**

بسم الله الرحمن الرحيم

Schistosomiasis

Dr. Abdulsalam Al-Akel

إعداد /

دائرة طب المجتمع – اللجنة العلمية 37A

في حال وجود أي سؤال أو استفسار يرجى التواصل معنا عن طريق بوت الدائرة

@Communitymedicine37ABot

Community Medicine Department

Faculty of Medicine and Health Sciences

Sana’a University

Dr. Abdulsalam Al-Akel

Objectives of the lecture

By the end of this lecture, you will be able to:

1. Know neglected tropical diseases
2. Mapping Epidemiological Burden of Schistosomiasis
3. Situation Analysis of Schistosomiasis in Yemen
4. Yemen National Schistosomiasis Control Program (NSCP)
5. Life Cycle of Schistosomiasis
6. Control an Prevention Strategies

➢ ADDITIONS …

❖Four diseases in Yamen that increase mortality and morbidity :

- 1-Respiratory infections .
- 2-diarrhea .
- 3- malaria .
- 4-schistosoma .

Neglected Tropical Diseases

Schistosomiasis

➢ ADDITIONS …

- العالم غير مهتم بهذا المرض لأنه لا يصيب إلا الدول النامية الفقيرة وليس منتشر عالمياً والإصابة به يؤدي إلى عبء اقتصادي .
- In all the word the first disease is acute respiratory syndrome (ARS) .

Neglected Tropical Diseases

1. Buruli Ulcer	11. African Sleeping Sickness
2. Chagas Disease	12. Leishmaniosis
3. Dengue Fever	13. Leprosy
4. Chikungunya	14. Lymphatic Filariasis
5. Dracunculiasis	15.Mycetoma
6. Echinococcosis	16. Onchocerciasis
7. Foodborne Trematodiases	17. Rabies
8. Soil-Transmitted Helminths	18. Scabies
9. Taeniasis	19. Schistosomiasis
10. Cysticercosis	20. Snakebite

➢ ADDITIONS ...

- يوجد ١٧٠ ألف حالة من داء الكلب في العاصمة صنعاء ، تم القضاء على ١١ ألف وتم التوقف لذا ازدادت الحالات والأطفال هم الأكثر عرضة .
- في ٢٠٠٩ م كان قد انتهى تواجد مرض شلل الأطفال والحصبة في اليمن لكن الأن رجعت الأمراض وحصلت انتكاسه .
- أيضاً ما يزال هناك الكثير من الأمراض المنتشرة المهملة في اليمن .
- يوجد أكثر من ٢٠ مرض في اليمن منتشر بسبب الماء الغير نظيف .

➢ ADDITIONS …

❖Why do we care about schistosomiasis ?!

➢ Because it is …..

- 1-very common .
- 2-Attack children because they swimming in pools .
- 3-Increase mortality and mobidity rate .
- 4- 3 million of child attack every years and 6 millions need to treatment.

Key Messages

- Schistosomiasis is considered a neglected tropical disease (NTD).
- Control efforts have long been in vain (ineffective).
- Interventions against schistosomiasis are escalating.
- Diagnosis before drug intervention was deemed unnecessary and not cost-effective.
- Praziquantel is safe, efficacious and inexpensive when delivered through the school platform.

Epidemiological Burden of Schistosomiasis

- Schistosomiasis is an acute and chronic parasitic disease
- It is caused by blood flukes (trematode worms) of the genus Schistosoma.
- Recent estimates indicate that 779 million people are at the risk of infection
- The disease is so common.
- The disease is most commonly found in Africa, Asia and South America.
- 85% of those in risk areas live in Africa.
- Approximately 207 million individuals in 78 countries are infected with schistosomiasis.
- 120 million of these people developed the disease.

Epidemiological Burden of Schistosomiasis

- In 2019, at least 236.6 million people required preventive treatment.
- An estimated 24,270 to 200,000 people die each year.
- In tropical countries, schistosomiasis is second only to malaria among parasitic diseases.
- It leads to a great economic impact.
- Preventive treatment, which should be repeated over a number of years, will reduce and prevent morbidity.
- 51 endemic countries require preventive chemotherapy for schistosomiasis, with moderate-to-high transmission.

Epidemiology of Schistosomiasis in Yemen

- The prevalence of **urinary schistosomiasis** was more than 80% (some areas of Amran, Hajjah, **Dhamar**, Sana'a and Rima).
- The prevalence of **intestinal schistosomiasis** was more than 70% (some areas of Taiz, Ibb and **Damar**).
- In some provinces, the two types of Schistosomiasis are found in varying proportions
    - **particularly** in the central regions of the country (Sana'a, **Dhamar** - Ibb).
- The prevalence of Schistosomiasis nationwide decreased to less than 10%.
    - **This was due to** the implementation of the World Bank-funded anti-Schistosomiasis project from 2010 to 2017).

Epidemiology of Schistosomiasis in Yemen

- Prevalence 19.4 % in 2012 and declined to 8.3% in 2014.
- Prevalence in some districts of some governorates.

Governorate	Prevalence %	Year
Taiz	37	1983
Amran	29	1989
Dhamar	23 (S.Mansoni) 17 (S.haematobium	1992

Epidemiology of Schistosomiasis in Yemen

- Both the urinary and the intestinal form of Schistosomiasis are endemic in Yemen
- Transmission is widespread.
- The disease is of high, meso, and low endemicity mostly in the western mountainous region
- 222 out of 333 districts are considered endemic or likely to be endemic.
- 3 million children and 6 million adults require treatment every year.
    - This figure is believed to be an underestimate of the actual disease burden.
    - The mountainous areas in the western part of the country are most severely affected.

National Schistosomiasis Control Program (NSCP)

- The first comprehensive control strategy was initiated in 2008
- The World Bank supported the strategy financially in 2009 for 6 years program
- The control strategy is based on preventative **chemotherapy.**
- Praziquantel (with albendazole or mebendazole) to target Soil-transmitted helminthiasis.
- Yemen’s control programs cover nearly 80% of those in need.
- Schistosomiasis treatment was distributed to 34% of the population.
- Since 2010, the National Schistosomiasis and Parasites Control Programme (NSCP) has conducted 14 campaigns.

National Schistosomiasis Control Program

- **The program distributed:**
    - around 92 million tablets of praziquantel, and
    - 30 million tablets of albendazole.
- The NSCP trained 88,000 team leaders and drug distributors.
- Active chemotherapy began in 2010.
- Program interrupted in 2011 due to civil and political unrest.
- Prevalence has been reduced from a baseline **of 19.4% to 6.9%.**
- WHO and the MoPHP and Yemen NSCP Initiative has developed a risk map of schistosomiasis prevalence in Yemen.
- This will improve estimates of the population infected and at risk.

Causes of schistosomiasis endemicity?

1. Poor water supply and sanitation services, especially in rural areas.
2. Agricultural expansion
3. Expansion of the construction of ponds, dams and water barriers.
4. Abnormal habits and behaviors of the population such as urination and defecation within or near water sources.
5. Weak role of health facilities in implementing various control activities.
6. Low level of health awareness among citizens about schistosomiasis and the factors helping to spread it.

➢ ADDITIONS

❖ يزداد الإصابة به عند الأطفال دون سن الخامسة "١٠٠ ضعف " لعدة أسباب أهمها :

- السباحة في المياه الراكدة . **"من أهم الأسباب "**
- لأنهم أكثر نشاطاً وحركه ببداية اكتشافهم للحياة .
- زيادة اتصالهم مع الناس .
- زيادة اتصالهم مع العوامل المسببة للمرض .

❖ ملاحظات :

❑ ٢٦ مليون من الناس لا يمتلكون مصدر نقي للمياه ، ٣١% لا يمتلكون لا مأوى ولا مسكن .

❑ قنوات التصريف مهمة قبل وجود السدود ، لماذا ؟

للحفاظ على مصدر المياه من التلوث حيث أن كل ٧٠٠ ألف بويضة تتطلق في المياه العذبة .

➢ Important

Criteria of Schistosomiasis Endemicity (NSCP)

Area	Criteria
High Prevalence Area	> 40% of either infection
Medium Prevalence Area	10 - 40% of either infection
Low Prevalence	<10% of either infection
Suspected Area	Currently unmapped

Schistosomiasis

- German physician Theodor Bilharz, the first to describe the cause of urinary schistosomiasis in 1851.
- Schistosomiasis is a fresh water-based parasitic disease.
- It is a disease caused by infection with parasitic blood flukes.
- Infection occurred when skin comes in contact with contaminated water
- The skin is penetrated by cercariae.
- The intermediate snail hosts for S. haematobium is Bulinus truncates
- The intermediate snail hosts for S. mansoni is Biomphalaria arabica

Biomphalaria Arabica

هو الملفوف

Bulinus Truncatus

هو المقطوع

http://urineanalysis.blogspot.com

➢ ADDITIONS

Eggs

- S. Mansoni
  - شوكتها طرفية .
  - تدخل لل GIT .

- S. Haematobium
  - شوكتها جانبية .
  - تعمل تخديش للمثانة ولهذا يظهر الدم في بول المصاب بها .

snails

Causes of Human schistosomiasis

1. Schistosoma mansoni (Africa and Nile River valley in Sudan and Egypt, Yemen, South America, Caribbean (risk is low): Dominican Republic, Guadeloupe, Martinique, and Saint Lucia).

2. S. haematobium (Africa, Nile River valley in Egypt, Yemen, North Africa in areas of the Middle East)

3. S. japonicum (Indonesia and parts of China and Southeast Asia)

4. S. mekongi (Cambodia and Laos)

5. S. intercalatum (parts of Central and West Africa).

- Other species of schistosomes, which parasitize birds and mammals, can cause cercarial dermatitis in humans.

Essential Features of Transmission of schistosomiasis

- **Fresh water**
  - slow moving to stagnant
  - edges of dams, rivers, canals, ponds
- Presence of appropriate snails
- Contamination of water with viable eggs
- Human contact with water containing cercariae

Vulnerable Population Groups

- Schistosomiasis mostly affects poor and rural communities
- Agricultural and river fishing populations are particularly affected.
- Women doing domestic works in infested water, such as washing clothes
- Women are also at risk and can develop female genital schistosomiasis.
- Inadequate hygiene and contact with infected water make children especially vulnerable to infection.
- Migration to urban areas and population movements are introducing the disease to new areas.

Infection and Transmission

- People become infected when larval forms of the parasite – released by freshwater snails – penetrate the skin during contact with infested water.
- Transmission occurs when people suffering from schistosomiasis contaminate freshwater sources with their excreta containing parasite eggs, which hatch in water.
- In the body, the larvae develop into adult schistosomes.
- Adult worms live in the blood vessels where the females release eggs.

Infection and Transmission

- Some of the eggs are passed out of the body in the faeces or urine to continue the parasite’s lifecycle.
- Others become trapped in body tissues, causing immune reactions and progressive damage to organs.

➢ ADDITIONS

❖Shedding of eggs to :

- Liver .
- Urinary bladder .

❖And may go to :

- Lung .
- Spinal cord .
- Skin .
- Brain .
- Appendix .
- Fallopian tube .
- Epididymis .

❖ Adults are not found in the water but they are found in :

- Mesenteric plexus .
- Portal plexus .
- Venous plexus .
- Azygous vein .

➢ ADDITIONS

❑58 millions of women are infertile by schistosomiasis .

❖ لماذا النساء أكثر عرضة للإصابة بالبلهارسيا ؟

• لأنها الأكثر استخداماً للمياه اكثر من الرجال .

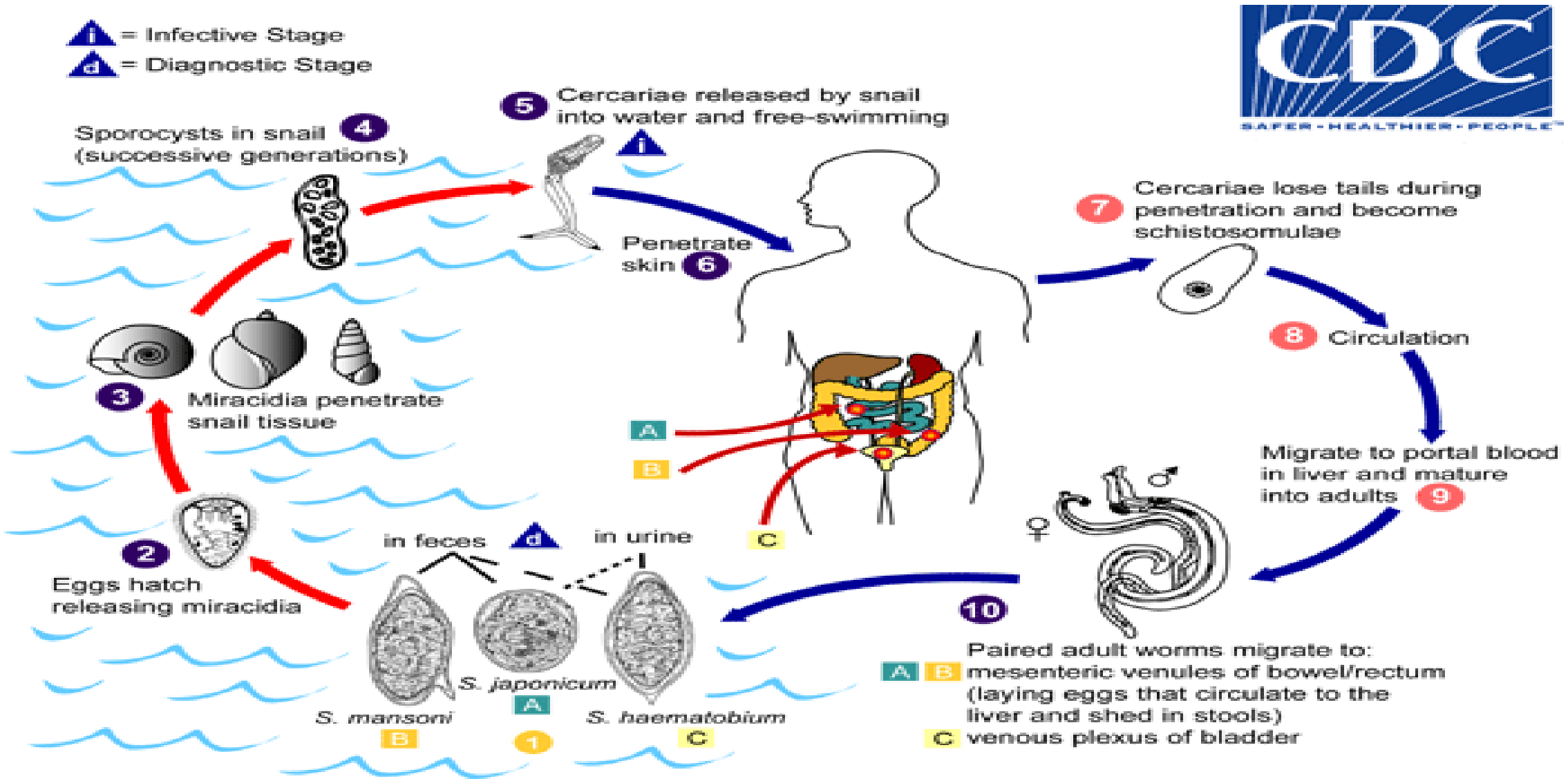
❖ هل ممكن الواحد يصاب بالبلهارسيا من ال mucous of mouth ؟

• نعم ، وذلك إذا استقر الماء في الفم أي أنه ضل لفترة داخل الفم ، أما إذا نزل للمعدة فإنها تموت بسبب حموضة المعدة acidity .

❖ من المهم أن نعرف عن دورة حياة البلهارسيا ، لماذا ؟

• من أجل معرفة كيف عمل لها control

Life Cycle of Schistosomiasis



Pathology of Schistosomiasis

- Most people have no symptoms when they are first infected.

Early stage - Initial penetration of skin by cercariae results in:

  - hypersensitivity and a transient pruritic rash
  - popular skin rash (swimmer's itch).

Intermediate stage

- After penetration, the organism enters the bloodstream and migrates through the lungs.
- Four to 8 weeks after exposure, an acute illness can develop

Pathology of Schistosomiasis

S. mansoni and S. japonicum schistosomiasis

- Katayama fever
- Abdominal pain
- Diarrhea
- Bloody stool
- Chills
- Cough
- Muscle aches
- Bleeding per rectum

Pathology of Schistosomiasis

Katayama fever is a manifestation of acute schistosomiasis.

Typical features of S. Mansoni include:

- Fever
- Malaise
- Abdominal pain, diarrhea
- Urticarial rash
- Enlarged liver and spleen
- Bronchospasm

Typical features of S. haematobium includes:

- Hematuria (painless and terminal)
- Urine obstruction
- Mass in the urinary bladder
- Scarring, Calcification of UB
- Squamous cell carcinoma of UB
- Occasional embolic egg granulomas in brain or spinal cord.

Common Complications of Schistosomiasis

Complications

Without treatment, schistosomiasis can persist for years.

- Liver damage (hepatic granuloma)
- Kidney failure
- Pulmonary hypertension (core pulmonale) due to eggs in lung
- Infertility
- Bladder cancer

Unusual Complications of Schistosomiasis

Brain: convulsion, encephalitis, coma

Spinal canal: acute myelopathy

Anywhere (appendix, peritoneum, skin)

➢ ADDITIONS

❖Schistosoma is characterized by :

- ➢ 1- Acute stage
  - Katayama fever .
  - Abdominal pain.
  - Hematuria .
  - Arthralgia .
  - Diarrhea .

Katayama

الاسم يعود إلى نهر في اليابان ، قبل ١٨٥١ م كان يعتقد عند اليابانيين أي شخص يسبح بهذا النهر يصاب بهذه الحمى بجانب أعراض الإصابة بالبلهارسيا ولهذا سميت هذه الحمى بهذا الاسم .

➢ ADDITIONS

➢ 2- chronic stage

- ❑ نتيجة للعلاج أحياناً يحصل لها استقرار داخل الجسم لمدة حوالي ٣٠ عاماً وتتحول إلى بلهارسيا مزمنة .
- ❑ إذا حصل مرض سبب ضعف للمناعة فإنها تؤدي إلى ظهور المضاعفات التالية :
  - Portal hypertension .
  - Cancer .
  - Squamous cell carcinoma .
  - Encephalitis .
  - Splenomegaly .
  - Infertility .

Economic Health Effects

- The economic and health effects of schistosomiasis are considerable, and the disease disables more than it kills.
- In children, schistosomiasis can cause anaemia, stunting and reduced ability to learn.
- The effects are usually reversible with treatment.
- Chronic schistosomiasis may affect people’s ability to work and, in some cases, can result in death.
- The number of deaths due to schistosomiasis is difficult to estimate because of hidden pathologies such as liver and kidney failure, bladder cancer and ectopic pregnancies due to female genital schistosomiasis

Diagnosis of Schistosomiasis

- Stool or urine samples can be examined microscopically for parasite eggs.
    - Stool for S. mansoni or S. japonicum eggs
    - Urine for S. haematobium eggs.
- The eggs tend to be passed intermittently and in small amounts, may not be detected.
- rapid diagnostic tests for antigen detection.
- polymerase chain reaction (PCR) assays.

➢ ADDITIONS

❖ Dignostic stage → eggs .

- But in chronic cases there are no eggs → so we will use ultra sound for urinary bladder & liver .

❖ Infective stage → cercaria .

Treatment of Schistosomiasis

1. The WHO has developed guidelines for community treatment.
2. The treatment is based on the impact the disease on children in villages:
    - When a village reports more than 50 percent of children have blood in their urine, everyone in the village receives treatment.
    - When 20 to 50 percent of children have bloody urine, only school-age children are treated.
    - When fewer than 20 percent of children have symptoms, mass treatment is not implemented.

Treatment of Schistosomiasis

Praziquantel is the recommended treatment against all forms of schistosomiasis.

It is effective, safe, and low-cost.

20 mg/kg PO/ TID for 1 day (at intervals 4-6 hr.)

➢ ADDITIONS

❖Why do we give praziquantel with albendazole or mebendazole ?!

- Due to protection of anaphylactic shock .
- Anaphylactic shock is caused by spread of eggs of any parasite in the body , so we give albendazole to kill the eggs of parasite specially ascaris .

- Praziquantelما ينفع وخاصة في حالة ال chronic لهذا يتم عمل له ultra sound للكبد والمثانة.
- إذا أنا شكيت بالhaematobium فإننا أعمل على تزويد الجرعة لل 25 mg لأن الامتصاص في ال GIT تكون عالية .

Control and Prevention of Schistosomiasis

Prevention: No vaccine is available.

The WHO strategy for schistosomiasis control focuses on reducing disease through periodic, targeted treatment with praziquantel through the large-scale treatment (preventive chemotherapy) of affected populations.

It involves regular treatment of all at-risk groups.

Control of Schistosomiasis

Strategy of control

The life cycle can be attacked at various sites

1. Contamination of water
2. Intermediate host
3. Human contact

Control of Schistosomiasis

1. Reducing sources of contaminated water

- Health education most important
- Sanitation improvement
- Prevention of access to transmission sites
- Reduction of eggs excretion (Chemotherapy)

2. Attack on intermediate host

- Permanent elimination of snail habitant by drainage and landfills
- Mollusides (temporary elimination) only effective in high controlled environment, may harm other species of animals
- It is costly, need to be repeated for indefinite times

Control of Schistosomiasis

3. Human contact with infection

- Provision of safe water supply
- Provision of latrines
- Reducing occupational contact through

Community participation is vital in the control process

Prevention of Schistosomiasis

Groups targeted for treatment are:

- School-aged children in endemic areas.
- Adults considered to be at risk in endemic areas
- People with occupations involving contact with infested water, such as:
    - fishermen, farmers, irrigation workers.
    - women whose domestic tasks bring them in contact with infested water.
- Entire communities living in highly endemic areas.

Prevention of Schistosomiasis

Prevention: No vaccine is available.

The best way to prevent schistosomiasis:

1. Avoid swimming or wading in freshwater in countries in which schistosomiasis occurs. تجنب السباحة أو الخوض في المياه العذبة في البلدان التي يحدث فيها البلهارسيا.
2. Swimming in the ocean and in chlorinated swimming pools is safe.
3. Bring drinking water to a rolling boil for at least 1 minute will kill any harmful parasites.
4. Drink safe water. rolling boil for 1 minute or filter water before drinking.

Prevention of Schistosomiasis

- Iodine treatment alone **WILL NOT GUARANTEE** water safety and free of all parasites.
- Water used for bathing should be brought to a rolling boil for 1 minute to kill any cercariae .
- Water held in a storage tank for at least 1 - 2 days should be safe for bathing.

➢ ADDITIONS

❖كيف يمكن حل مشكلة انتشار أي مرض ؟

ذلك عن طريق التالي :

- ١- تحديد ووضع أولويات للمشكلة الصحية في المجتمع عن طريق التنسيق بين مختلف القطاعات ، ثم اختيار المشكلة الأخطر منها .
- ٢- مشاركة المجتمع في التخطيط .
- ٣- المشاركة في عملية التمويل .
- ٤- المشاركة في عملية التنفيذ .

Challenges Ahead of Control of Schistosomiasis

1. Yemen is one of the poorest countries in the Arab world.
2. One of the fastest growing populations in the world.
3. Widespread poverty.
4. Increased level of food and water insecurity.
5. Lack of sanitation facilities and safe water.
6. Increasing snail habitat due to habitat modification.
7. Weak governance and institutional structure to deliver proper control measures.

➢ ADDITIONS

معلومات إضافية

- في عام ٢٠٠٠م كانت نسبة الإصابة بالملاريا في الحديدة ٤٨% وبعد عمل حملات لمكافحة الملاريا انخفضت نسبة الإصابة إلى ٣% في عام ٢٠٠٨ م.

- أما بالنسبة لسقطرى فقد كانت في عام ٢٠٠٠م نسبة الإصابة فيها ١٠٠% وبعد عمل حملات المكافحة انخفضت نسبة الإصابة إلى ٠% ، **لماذا ؟**
  - وذلك بسبب أنها مدينة محصورة ، عدد سكانها قليل ، ممنوع الدخول إليها ، والبيئة متجانسة لذلك حصل استئصال للملاريا .

➢ ADDITIONS

معلومات إضافية :

❑ معدل استهلاك الفرد للمياه سنوية :

- يستهلك الفرد حوالي ١٢٥٠ لتر مكعب من الماء سنوياً في العالم .
- يستهلك الفرد حوالي ٦١ لتر مكعب من الماء سنوياً في الشرق الأوسط .
- يستهلك الفرد حوالي ٦١ لتر مكعب من الماء سنوياً في اليمن .

❖ لهذا تعد اليمن بلدة شحيحة بالمياه وتنتشر بها مختلف الأمراض ... .

Iodine Deficiency Disorders (IDDs)

By: Dr. Khaled Algendari

Iodine deficiency:

Iodine deficiency is recognized as global problem. An estimated population of 1 billion is at risk, because they live in environments where the soil has been deprived of iodine. In the at-risk global population Health Organization estimated that in excess of 200 million have goiter and 20 million have some degree of brain damage caused by the effect of iodine deficiency in pregnancy. Surveys in many parts of the world have demonstrated that endemic goiter preferentially affects rural population and low income groups living under poor sanitary conditions.

In Yemen, the Total goiter rate (TGR) estimated in 1998 among 16.8% of school age children, and 17.3% µg/d as urine iodine excretion.

What is iodine?

It is a trace mineral essential for human survival, and an integral part of the thyroid hormones of thyroid gland.

Why do we need iodine?

- For growth, development and reproduction, iodine is importance for the development and function of the brain and nervous system. As well as for maintenance of body temperature.

Sources of iodine:

- Iodine occurs in extremely variable amounts in food and drinking water.
- Iodine comes from the soil if soil contains enough iodine plant foods grown on it contain enough iodine.
- Sea foods and all salt water fish are rich sources, fresh water fish contain lower amounts but could be considered as potent sources.
- Amount of iodine in other animal foods depend on the iodine in the foods that they eat.
- Iodine also enters the food chain through the use of iodine compounds as disinfectants, and coloring agents.

What are the effects of iodine deficiency (ID)?

The damage done by lack of thyroid hormone varies in severity depending on the time in life at which the deficiency occurs.

The following table shows the different disorders associated with ID at different life stage.

Life stage	Major disorders
Fetus	Abortion, stillbirth, congenital anomalies, increased prenatal and infant mortality, psychomotor defects and cretinism in various degrees.
Neonate	Neonatal goiter and hypothyroidism.
Child and adolescent	Goiter, retarded physical development impaired mental function and intellectual performance.
adult	Goiter and impaired mental function. Hypothyroidism (the person feels cold easily, moves slowly, thinks slowly and appears unconcerned, may be sleepy, has a dry skin and may be constipated).

What can be done to prevent and treat IDDs?

- All the consequences of IDDs can be simply prevented by ensuring that everyone, especially girls, women of child bearing age and young children, consumes an adequate amount of iodine.
- There are several ways to give iodine:
  - Adding iodine to salt (iodized salt). It is the best way to obtain an adequate intake of iodine.
  - Giving large doses of iodine by mouth or by injection (iodized oil). It's a useful short-term method for areas of severe deficiency.

- Adding iodine to drinking water. It can be use in schools and household where iodized salt is not yet available and IDDs are a public health problem.

In Yemen, universal salt iodization program started by the year 1996

Care in use of iodized salt

1) Cooking reduced the iodine content of foods and iodized salt, so it is preferable to add salt by the end of cooking.
2) Salt should be kept in a tightly closed container to be protected from humidity.
3) Salt should be store in the kitchen away from sunlight and heat.

Methods of assessing IDDs:

- ❖ Examining for the presence and size of goiter. This is usually done by looking at the neck and palpating (feeling) the thyroid gland, or by Ultrasonography.
- ❖ Measuring the level of iodine in the urine. This must be done in a laboratory.
- ❖ Measuring the level in the blood of the thyroid stimulation hormone (TSH), this also must be done in a laboratory.

Classify the goiter:

Grade 0: There is No palpable or visible goiter.

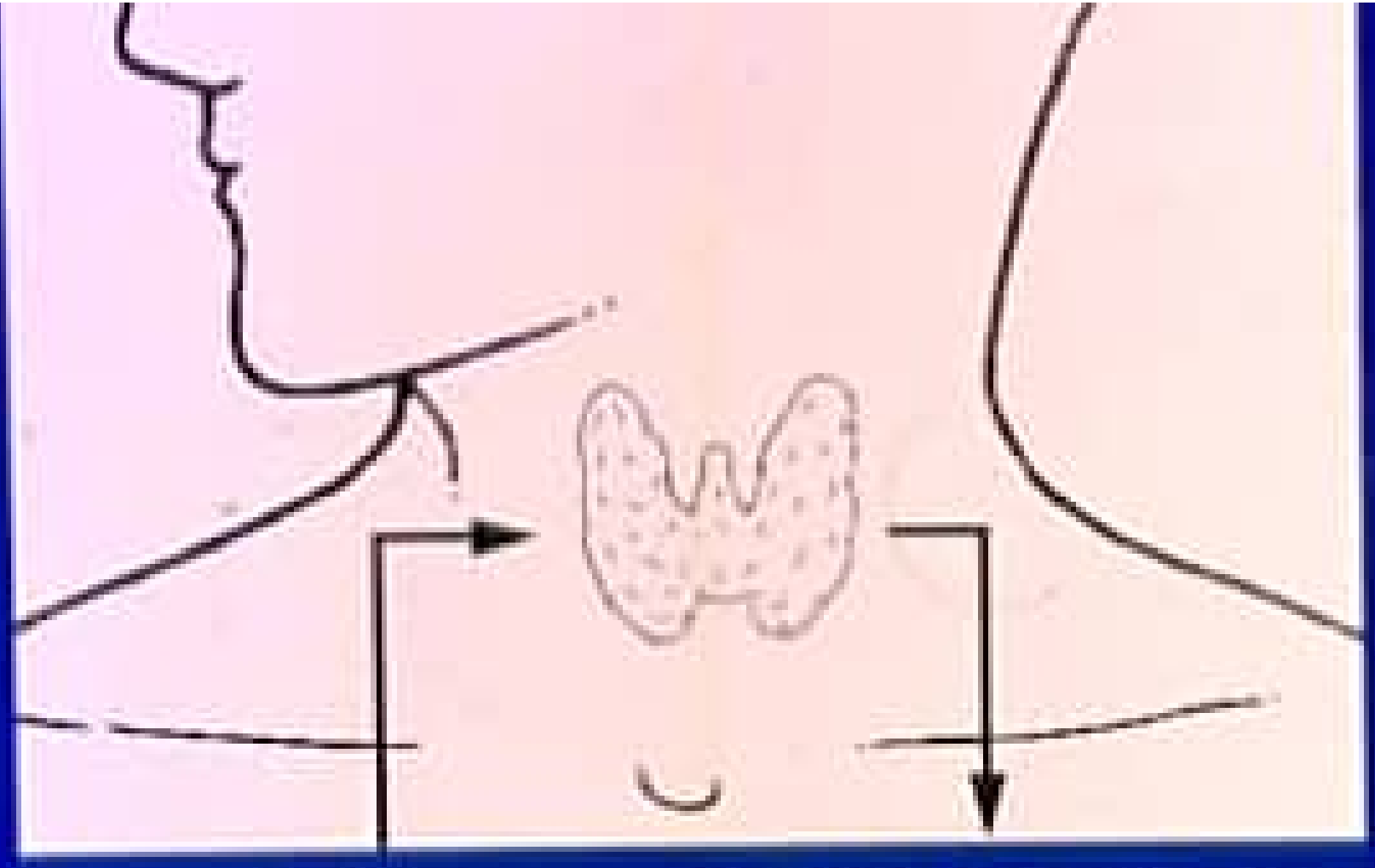
Grade 1: palpable goiter (goiter you can feel but cannot see with the head in the normal position. It moves upward in the neck when the person swallows).

Grade 2: There is Visible goiter (a swelling you can see with person's

head in normal position and which feels like a goiter when you palpate the thyroid gland).

Prevalence Indicators and criteria for public health problem for Iodine deficiency

Indicator	Mild	Moderate	Severe
Goiter > 0	5.0 – 19.9 %	20.0 – 29.9%	≥ 30.0 %
Median Urinary iodine level (µg/ dl).	5.0 – 9.9 %	2.0 – 4.9 %	< 2.0 %
TSH >5 m/u/1 whole blood	3.0 – 19.9 %	20.0 – 39.9 %	≥ 40.0%



Iodine

Thyroid hormones

- control production of heat and energy
- needed for development of the brain and nervous system

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Ways to control IDDs

- Add iodine to salt (salt iodization)
- Give high doses of iodine in oil by mouth or injection
- Add iodine to drinking water

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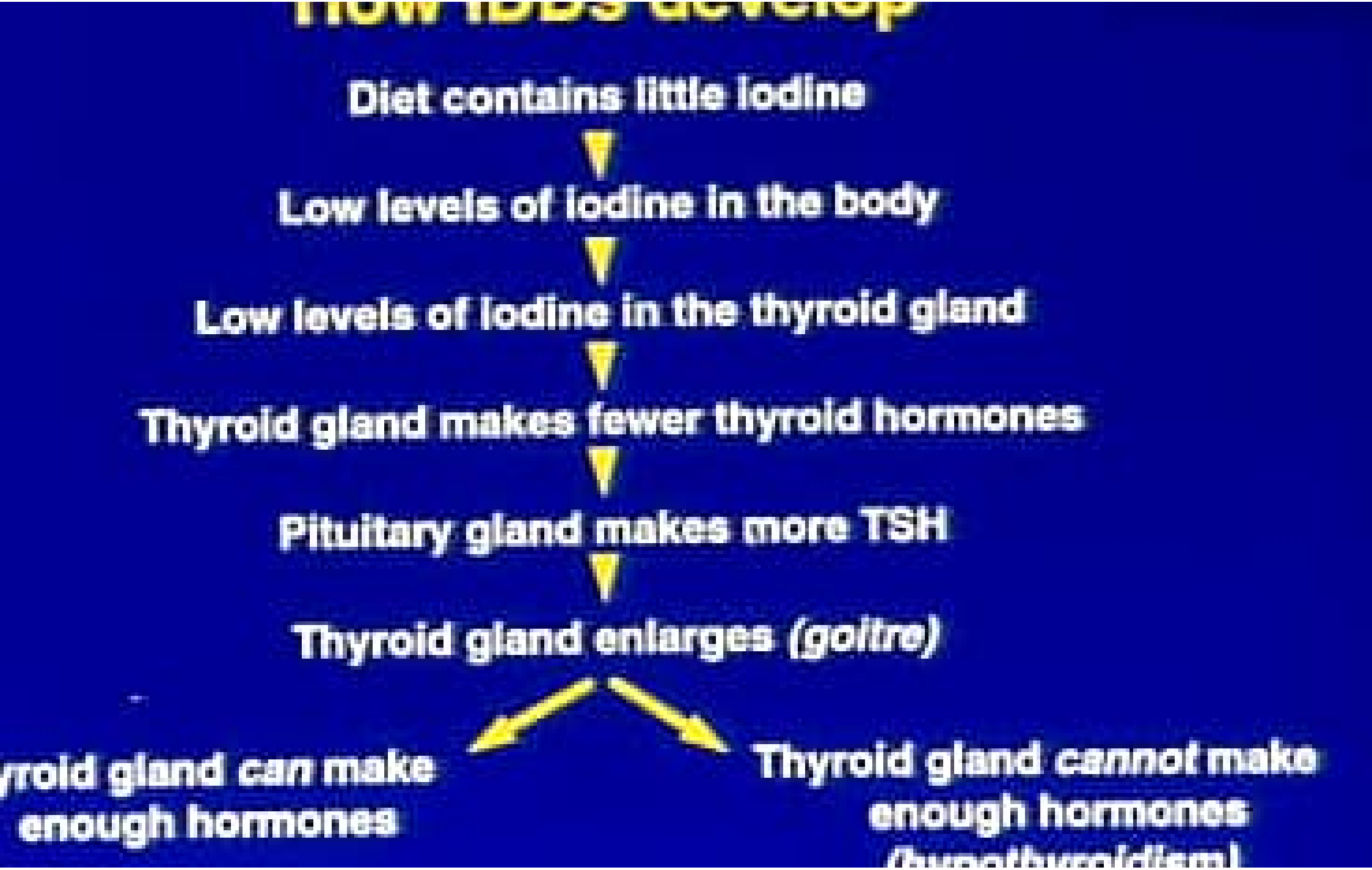

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Period at which different IDDs may develop

etus	Neurological and/or hypothyroid cretinism; other mental and physical defects; Increased risk of abortion or stillbirth or death around the time of birth
wborn baby	Hypothyroid cretinism; goitre; Increased risk of death
ld/adolescent	Goitre; hypothyroidism (Impaired mental function)
ult	Goitre and its complications; hypo-thyroidism (Impaired mental function)

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Ways to control IDDs

- Add iodine to salt (salt iodization)
- Give high doses of iodine in oil by mouth or injection
- Add iodine to drinking water

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Simple classification of goitre

Grade 0	No goitre
Grade 1	Palpable goitre
Grade 2	Visible goitre

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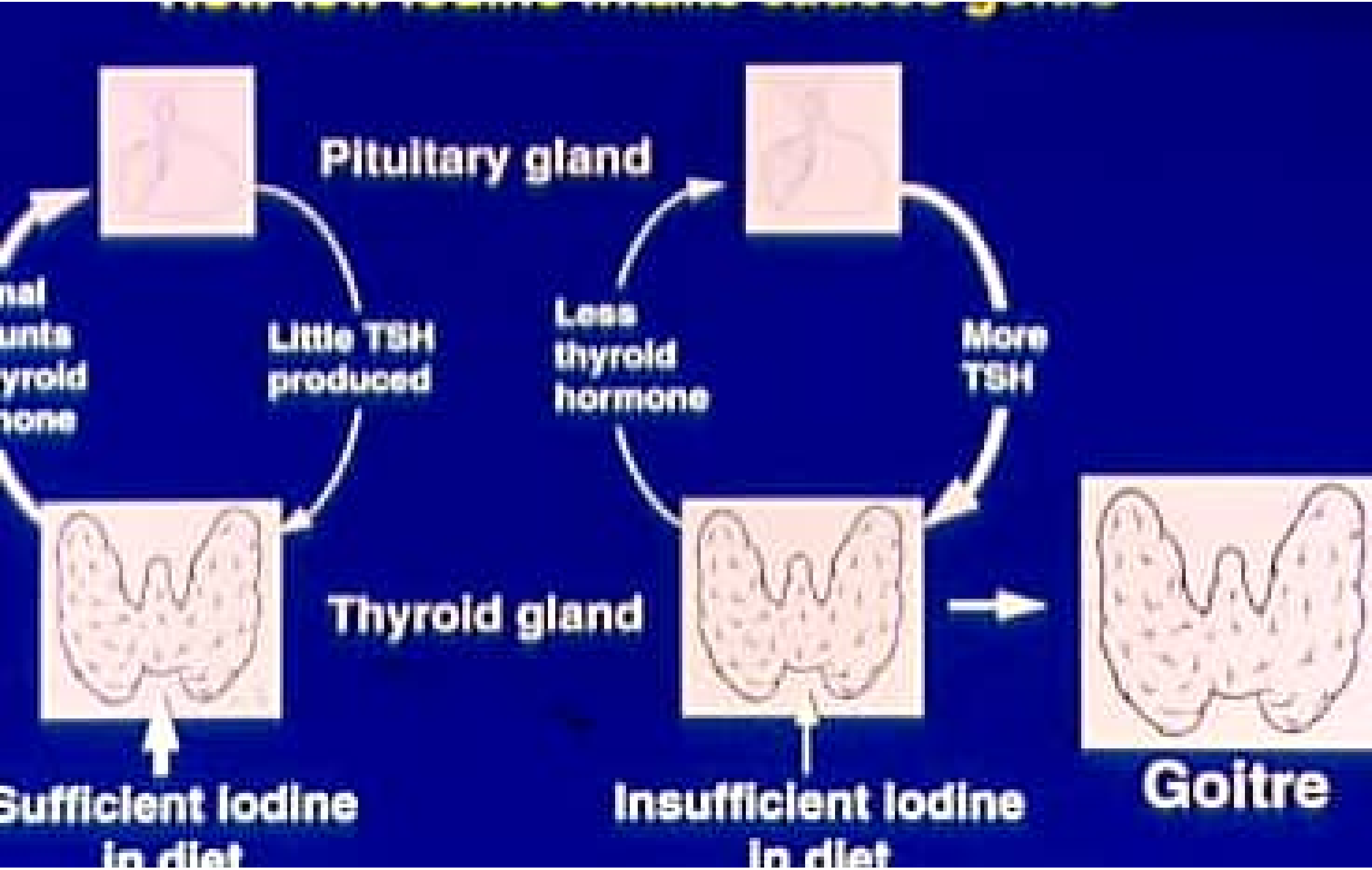

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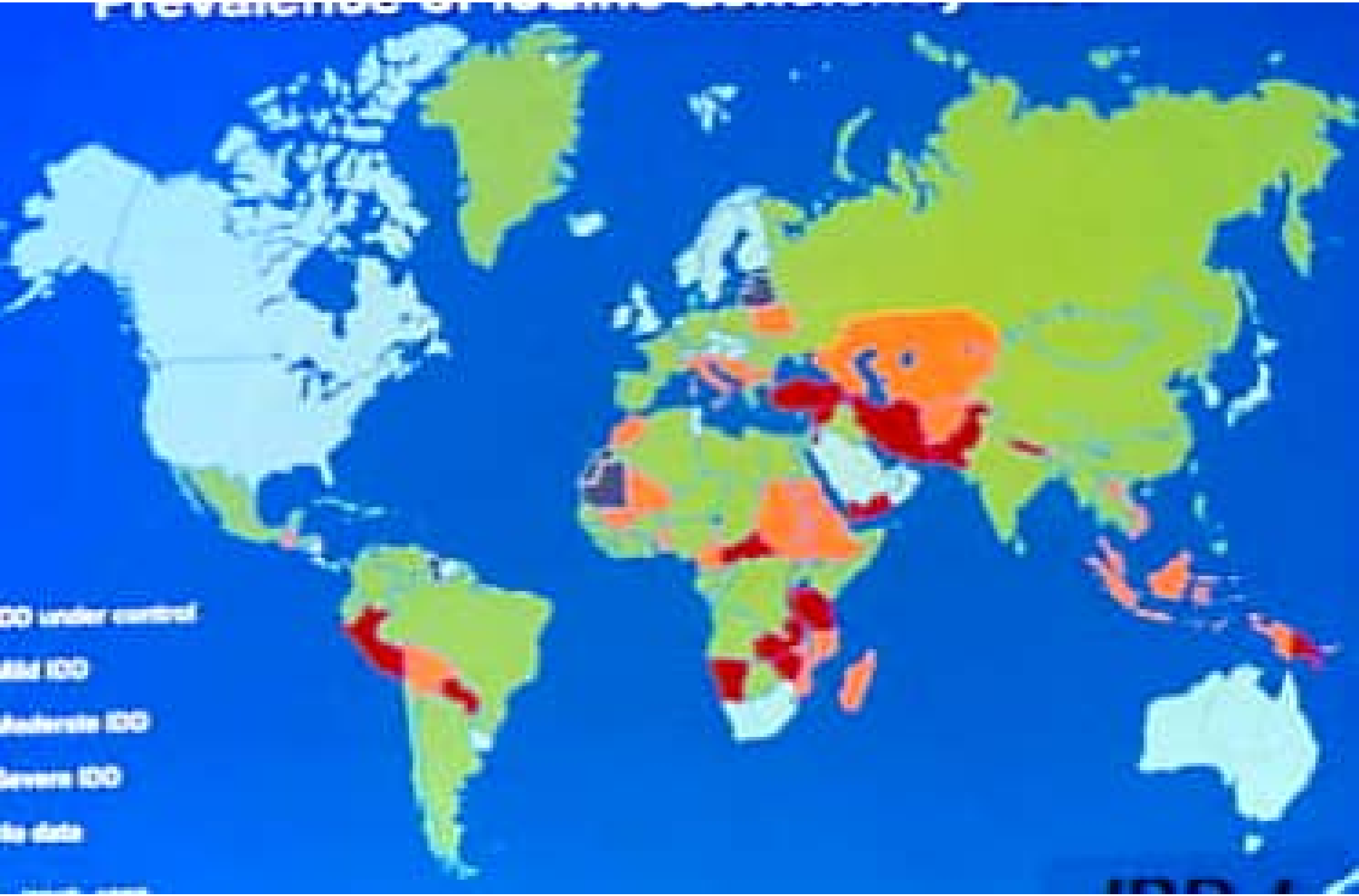

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Assessing IDDs

- Goitre
- Iodine in urine
- TSH in blood

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Sexually transmitted Infections(STI)

DR Kawkab Al-haddad

Definition

The sexually transmitted diseases (STD): are a group of communicable diseases that are transmitted predominantly by sexual contact (vaginal, oral, anal) and caused by a wide range of bacterial, viral, protozoal & fungal agents and ectoparasites.

There are more than 30 known bacteria, viruses & parasites that cause STIs.

Dramatic transformation of STD

First, the change in name from venereal diseases (V.D.) to sexually transmitted diseases (STD) indicates this transformation. The list of pathogens which are sexually transmissible has expanded from the 5 "classical" venereal diseases (syphilis, gonorrhoea, chancroid, lymphogranuloma venereum and donovanosis) to include more than 30 agents .

Classification of STI agents

Bacterial agents:

1. Neisseria gonorrhoeae
2. Chlamydia trachomatis
3. Treponema pallidum
4. Haemophllus ducreyi
5. Mycoplasma hominis
6. Ureaplasma urealyticum
7. Calymmatobacterium granulomatis
8. Shigella spp.
9. Campylobacter spp.
10. Group B streptococcus
11. Bacterial vaginosis-associated organisms

Viral agent

1. Human (alpha) herpesvirus 1 or 2 (herpes simplex virus)
2. Human (beta) herpesvirus 5 (formerly cytomegalovirus)
3. Hepatitis virus B
4. Human papilloma viruses
5. Molluscum contagiosum virus
6. Human immunodeficiency virus

Classification of STI agents

Protozoal agents

1. Entamoeba histolytica
2. Giardia lamblia
3. Trichomonas vaginalis

Fungal agents

1. Candida albicans

Ectoparasites

1. Phthirus pubis
2. Sarcoptes scabiei

Dramatic transformation of STD

Secondly, attention is now given not only to specific diseases, but also to **clinical syndromes** associated with STDs. Most of the recently recognized STDs are now referred to as second generation STDs. AIDS, the most recently recognized, is a totally new disease.

Common Syndromes and sequelae for w sexual transmission of epidemiological importance

- Male urtheritis
- Lower genital syndrome in women (vaginitis, cervicitis, urtheritis)
- Genital ulceration
- Proctitis/colitis
- Salpingitis
- Epididymitis/orchitis
- Infertility /ectopic pregnancy
- Perinatal and postnatal mortality
- Hepatitis /Hepatic carcinoma
- Genital carcinoma
- Acquired immunodeficiency syndrome

Extent of the problem

The true incidence of STDs will never be known because of :

1. **Inadequate reporting**
2. **Secrecy that surrounds them. All available data indicate a very high prevalence of STD.**

- **STDs have a profound impact on sexual and RH**
- **Rank among the top 5 disease categories for which adults seek health care.**

Extent of the problem

- More than 1 million STIs are acquired every day.
- 357 million new infections with 1 of 4 STIs each year (estimated):
- chlamydia (131 million)
- gonorrhoea (78 million)
- syphilis (5.6 million) and
- trichomoniasi.s (143 million).
- > 500 million people are living with genital HSV (herpes) infection. At any point in time,
- > 290 million women have an HPV infection

STI serious consequences.

- STIs like herpes and syphilis can increase the risk of HIV acquisition three fold or more.
- Mother-to-child transmission of STI can result:

1. stillbirth
2. neonatal death
3. low-birth-weight and
4. prematurity
5. sepsis,
6. pneumonia

STI serious consequences

- Over 900,000 pregnant women were infected with syphilis resulting in approximately 350,000 adverse birth outcomes including stillbirth in 2012.
- HPV infection causes about 528,000 cases of cervical cancer and 266,000 cervical cancer deaths each year.
- STI s such as gonorrhoea and Chlamydia are major causes of pelvic inflammatory disease (PID) and infertility in women .

STI in Yemen

???

Epidemiological determinants

Agent factors

Pathogen	Diseases
Neisseria gonorrhoeae	Gonorrhoea, urethritis, cervicitis, epididymitis, salpingitis, PID, neonatal conjunctivitis
Treponema pallidum	Syphilis
Haemophilus ducreyi	Chancroid
Chlamydia trachomatis	LGV , urethritis, cervicitis, proctitis, epididymitis, infant pneumonia, Reiter's syndrome, PIO, neonatal conjunctivitis
Calymmatobacterium granulomatis	Donovanosis (granuloma inguinale)
Herpes simplex virus	Genital herpes
Hepatitis B virus	Acute and chronic hepatitis
Human papillomaviruses	Genital and anal warts
Human immunodeficiency virus (HIV)	AIDS
Molluscum contagiosum	Genital molluscum contagiosum
Candida albicans &Trichomonas vaginalis	Vaginitis

Host factors

- The highest rates of incidence are observed in :
- 20-24 year-olds, followed by
- the 25- 29 and
- 15-19 years age groups.
- The most serious morbidity is observed during foetal development and in the neonate

Host factors

- The overall morbidity rate is higher for men than for women
- but the morbidity caused by infection is generally much more severe in women, e.g , PID.
- The frequency of STD infection is higher among single, divorced and separated persons than among married couples.
- Individuals from the lowest socio-economic groups have the highest morbidity rate.

Demographic factors

Factors contribute to increase in STDs in the developing countries:

- **population explosion and**
- **marked increase in the number of young people(group at highest risk for STD in the population)**
- **rural to urban migration**
- **increasing educational opportunities for women delaying their marriage and increasing STD risks.**

Social factors

1. Prostitution : This is a major factor in the spread of STDs. The prostitute acts as a reservoir of infection.

In Asia, most STDs are contracted from prostitutes.

- **Broken homes : Social studies indicate the promiscuous women are usually drawn from broken homes (unhappy)**
- **search of happiness outside home .**

Social factors

2. Changing behavioural patterns:

Conflict between chastity and independence, freedom from supervision, and equal rights for both sexes.

- **There has been a relaxation of moral& religion and cultural values in present-day society.**
- **The tendency to break away from traditional ways of life is particularly marked among young people.**

Social factors

3. Sexual disharmony : Married people with strained relations, divorced and separated persons are often victims of STDs.

4. Easy money : In most of the developing world, prostitution is simply a reflection of poverty. It provides an occupation for earning easy money.

5. Emotional immaturity : This has been often stressed as a social factor in acquiring STDs.

Social factors

6. Social stigma

- **non-detection of cases**
- **not disclosing the sources of contact**
- **dropping out before treatment is complete**
- **going to quacks for treatment**
- **self-treatment.**

Social factors

7. Social disruption

- **caused by disaster , war, civil unrest have always caused the spread of STD**

8. International travelers:

- **Travelers can import as well as export infection**
- **Travelers have important role in spread of STD**

Social factors

e.g of the role of international travelers in spread of STD is th rapid spread of the resistant strains of N. gonorrhea and AIDS throughout of the world

9. Alcoholism : The effect of alcohol seems to be more indirect than direct. Alcohol may encourage prostitution and conversely, prostitution may boost the sale of alcohol.

Basic Principles and STD Practices

1. ***Maintenance of confidentiality*:**

- **is of utmost importance.**
- **patient's details must not be disclosed to other patients or even other healthcare professionals without the patient's informed consent.**

Basic Principles and STD Practices

***2. Provision of information*: The patients should be provided with**

- **clear and accurate written information pertaining to their conditions & long-term implications of the condition for themselves as well as their partner.**
- **patients with STIs should be advised not to have sexual intercourse until their TTT as well as the TTT& follow-up of partner has been completed.**

Basic Principles and STD Practices

2. **Screening for other STIs**

- **Patient with one STI may be at risk of other STIs also.**
- **Before performing these tests, informed consent must be taken from the patient after explaining her about nature of the procedure.**
- **follow-up procedure in case the test comes out as positive.**

Basic Principles and STD Practices

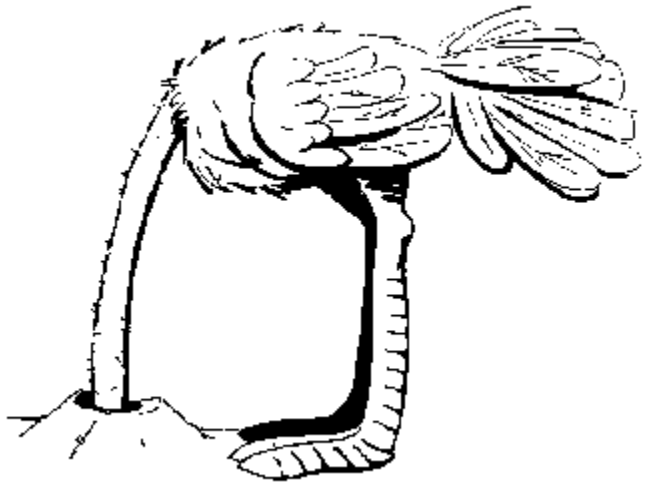
٣. Partner notification

- **The patients inform their partners about the risk of STI.**
- **This term has now replaced the term, 'contact tracing'.**
- **Sexual contacts of a patient with an STI are informed that they may be at risk, following which they are then offered screening, and treatment if indicated.**

Basic Principles and STD Practices

4. Contact tracing

This is a process through which the health workers directly contact the sexual contacts on the index patient with STI and then manage them appropriately.



Don't hide your head in the sand!

Gonorrhoea

- Greek words ‘gonos’ (seed) and ‘rhoia’ (flow) implying ‘flow of seeds’ and is caused by the bacterium *N. gonorrhoeae*.
- Gonorrhoea is spread through contact with the penis, vagina, mouth, or anus. It can also be spread from mother to baby at the time of delivery (vertical transmission) cause severe conjunctivitis, also known as ophthalmia neonatorum.
- Gonococci usually penetrate the columnar epithelial cells, the stratified squamous epithelium is relatively resistant to infection

Gonorrhoe

- cervix and rectum in women: Primary site of infection endocervix (columnar epithelium), then infection can spread to urethra and vagina giving rise to a mucopurulent discharge.
- urethra in men and women
- the rectum in men who have sex with men.
- the throat (pharyngitis) and the eyes.
- The incubation period is 2–8 days.

Clinical Presentation

- acute urethritis resulting in dysuria and a purulent penile discharge.
- may extend along the urethra to the prostate, seminal vesicles and epididymis, resulting in complications such as epididymitis, prostatitis, periurethral abscesses, and chronic urethritis.
- The infection may spread to the periurethral tissues, resulting in formation of abscesses and multiple discharging sinuses

Clinical Presentation

- mucopurulent vaginal & uretheral discharge.
- dysuria, and
- abdominal pain.
- Gonorrhoea can rarely cause intermenstrual bleeding or menorrhagia as a result of endometritis.
- Acute salpingitis may be followed by PID and infertility

Investigations

1. **NAAT Nucleic acid amplification tests: These tests show higher sensitivity than culture in both symptomatic and asymptomatic cases of gonorrhoea.**
2. **Culture and sensitivity of endocervical and urethral swab specimen Patients in whom NAAT is positive must be confirmed for gonorrhoea by culture and sensitivity test**

Treatment

Ceftriaxone 500 mg IM

Or

cefixime 400 mg (oral if IM dose is contraindicated or refused)

or

spectinomycin 2 g IM(as a single dose).

+ Doxycycline 100 mg BID for 7 days

or

azithromycin1 g PO (single dose)

Traetment Pregnant or Breastfeeding Mothers

- Ceftriaxone 500 mg IM single dose or spectinomycin 2 g IM as a single dose

+ Erythromycine 500mg four times for 7 days or Amoxicillin 500mg 3times for 7 days .

- Use of quinolone (like ofloxacin-ciprofluxacine) or tetracycline& Doxycycline antibiotics are usually not advisable during pregnancy.
- Use of azithromycin is advisable only under circumstances where adequate alternatives are not available.

Chlamydial Infection

Aetiology :

***Chlamydial infection caused by Chlamydia trachomatis which* is**

- **Gram-negative**
- **Aerobic**
- **Intracellular pathogen**
- **Coccoid or rod shaped.**
- **Different from other bacteria because it requires growing cells in order to remain viable.**

Clinical presentation :

- Asymptomatic.
- Vginal discharge, dysuria, abdominal pain, increased urinary frequency, urgency, urethritis, and cervicitis.
- PID: Chlamydia is very destructive to the fallopian tubes. fever, pelvic cramping, abdominal pain, or dyspareunia.
- Infertility or even absolute sterility due to PID
- Increased incidence of tubal pregnancy: Tubal destruction

Chlamydia & gonorrhoea

- PID associated with higher rates of consequent tubal infertility, ectopic pregnancy, and chronic pelvic pain in comparison with PID caused by other infections such as gonorrhoea.
- patients with may develop an inflammation of the liver capsule and adjacent peritoneal surfaces resulting in perihepatitis. This is also known as Fitz-Hugh–Curtis syndrome. Neisseria gonorrhoeae not only causes similar clinical syndromes as C. trachomatis but also coexists in a significant percentage of patients with chlamydial infection. **Testing for C. trachomatis should also be simultaneously tested for N. gonorrhoeae.**

Investigation

- ***Nucleic acid amplification testing***: using either polymerase chain reaction (PCR) or transcription-mediated amplification (TMA) of vulvovaginal swabs in women. Test of choice for establishing the diagnosis of chlamydial infection.
- **First-catch urine samples and endocervical swabs** (if available) can also be used.
- If amplification techniques are unavailable, tests such as antigen detection and genetic probe methods can be applied to endocervical or urethral swabs for diagnosing chlamydial infection.

Investigation

- ***Polymerase and ligase chain reactions*: Newer tests such as polymerase and ligase chain reactions, DNA probe, and DNA amplification are also being used.**
- ***Immunoassay*: Rapid tests for chlamydia may be used for establishing the diagnosis in low-resource settings.**
- **Enzyme-linked immunosorbent assay (ELISA) is no longer recommended in the UK.**

Prevention

- Early DX and TTT of asymptomatic infection (to reduce the transmission & complication).
- All sexually active individuals > 25 years are educated about chlamydia, and provided access to sexual health services to be annually screened .
- Screening for chlamydia should be offered as an integrated component of the existing sexual and reproductive health services including primary care-based services, community contraceptive services etc

Prevention

- internet-based testing and pharmacy-based testing to ensure that young people have universal access to testing.
- home-based methods of screening involving the self-collected first-void urine specimen and
- vulvovaginal swabs have been found to be acceptable.
- If an individual test is +ve for chlamydia, testing should be done again at 3 m following treatment.

Treatment

- azithromycin in a single dosage of 1 g per orally.

  Or

- doxycycline can be used orally 100 mg BID for 7 days.

  Or

- The combination of cefoxitin and ceftriaxone with doxycycline or tetracycline
- During pregnancy Erythromycin or amoxicillin in TID or QID dosage.

Treatment

- **Use of condoms often helps to prevent the spread of the infection.**
- **all newborns born to women infected with *C. trachomatis* must be treated with eye drops containing a broad-spectrum antibiotic (e.g., tetracycline), which kills chlamydia.**
- **follow-up, 6 weeks following the completion of treatment to check for partner notification, reinforcing health education, assessing the efficacy of treatment and exclusion of reinfection**

Complications

1. Infertility or even absolute sterility.
2. Tubal pregnancy.
3. Increased incidence of premature births.
4. Neonatal conjunctivitis or ophthalmia neonatorum
5. Pneumonia in the infant.
6. Increased risk of uterine infection in the mother

Genital herpes

- Genital herpes is a viral infection caused by the herpes simplex virus (HSV) (most commonly HSV II) .
- transmitted through sexual contact.
- HSV I is commonly responsible for causing herpes blisters in perioral region (orolabial herpes).
- HSV II is more commonly associated with lesions in the genital or the perianal area.

Aetiology

- Infection due to herpes could be either primary or recurrent (due to reactivation of the latent primary infection).
- Primary genital herpes infection spreads only by direct contact.
- Virus enters through the mucous membrane of the genital tract via microscopic tears.
- Virus travels to the nerve roots near the spinal cord and settles down permanently.
- Virus becomes latent and get periodically reactivated resulting in either symptomatic or asymptomatic infection with viral shedding.

- Incubation period, which generally lasts from 3 to 7 days before development of lesions begins. Prior to this, there are **no symptoms and the virus cannot be transmitted to others.**
- From the beginning of itching, until the time of complete healing of the ulcer, the **infection is definitely contagious**

Clinical Presentation

- Multiple painful blisters and **vesicles** are present on the external genitalia including the vulva, vagina, cervix, perianal area, or inner thigh,
- which ultimately develop into shallow and **painful ulcers** within a period of 2–6 weeks. They are frequently accompanied by **itching** and mucoid **vaginal discharge**. There may be associated **vulval pain**.

Clinical Presentation

- In case of **primary infection**, symptoms can be ***very severe***, resulting in swelling, ulceration, and infection of the vulva. Ulceration may also occur in the cervix.
- Primary infection may also be associated with **tender inguinal lymphadenopathy**, which is commonly due to secondary infection
- self-limited and usually heals without scarring.
- **Recurrent infection**, on the other hand, **is less severe** and self-limiting, and occasionally may be even asymptomatic.

Investigation

- ***Cytological tests***: **blister fluid sent to the laboratory in the viral culture medium. However, this test is not very sensitive(50% false-ve)**
- **PCR and HSV NAAT screening tests: used to identify HSV associated with higher sensitivity in comparison to the viral culture.**
- ***Immunological tests***: **blood tests for detecting antibodies**
- ***Biopsy***: **Tzanck smear is a rapid, fairly sensitive, and inexpensive method for diagnosing HSV infection. Smears are preferably prepared from the base of the lesions**

Treatment & prevention

- Treatment of genital herpes helps in shortening the duration of attack, preventing the occurrence of complications and reducing the risk of transmission
- Antiviral medication is usually recommended. Though the use of these medications can significantly help in reducing pain and decreasing the length of sores healing , treatment of the first infection **does not provide protection against future episodes**

Treatment & prevention

- patient not to touch her eyes or mouth after touching the blisters or ulcers.(Herpes can be spread from one part of the body to another during an outbreak).
- wash her hand scrupulously.
- Avoid sharing clothing that comes in contact with ulcers.
- Abstain from intercourse.
- Should always use condoms if a partner is infected

Treatment & prevention

1. ***General measures***:

- drinking large quantity of water to dilute the urine, thereby reducing pain on micturition
- measures which help in reducing pain include use of :
- saline bath
- analgesic drugs such as nonsteroidal anti-inflammatory drugs (NSAIDs),
- use of topical anaesthetic gel.

Treatment & prevention

2. Oral antiviral medications: All these drugs help in reducing **the severity and duration** of episodes. However, they do not alter the natural history of the disease

- aciclovir (200 mg five times a day),
- Famciclovir (250 mg three times a day) or
- valaciclovir (500 mg twice daily)

Complications

- ***Urinary retention*:**

may occur due to autonomic neuropathy or due to local reaction around the vulva and urethra because of severe vulval pain. Suprapubic catheterisation may be required in these cases.

- ***Post herpetic neuralgia*: This may be the cause of chronic vulval pain.**

Human Papilloma Virus

- Human papilloma virus (HPV) causes ano-genital **warts**, which vary from the common soft, flesh-coloured protuberances which may become exuberant (cauliflower like) to papular flat warts on drier a reas (eg. shaft of penis).
- They can be seen anywhere in the genitalia including in the perianal region.
- The other commonly recognized manifestation of genital HPV infection is **cervical cancer**, caused by some sub-types of HPV.

Human Papilloma Virus

- For any viral infection the mainstay of control is prevention, especially in young sexually active individuals(HPV vaccine)
- Regular examination of the cervix& cervical cytology using Pap smear is recommended for female patients and female contacts in order to detect progression of CIN lesions to cevical cancer.
- CCS and colposcopy services decrease incidence & mortality

Syphilis

- **Syphilis is an STD caused by the spirochete *Treponemapallidum*.**
- **Syphilis infection could be acquired or congenital.**
- **Though the route of transmission of acquired syphilis is almost always through sexual contact,**
- **sometimes congenital syphilis can occur via transmission from mother to child in utero.**

Clinical presentation

- Early stage
- late stage of infection.
- **Early infection** is typically characterised by three phases:

1. primary,
2. secondary,
3. early latent phase (<2 years of infection).

- **Late stage of infection** includes :

1. late latent phase (>2 years of infection)
2. Tertiary phase

Primary phase (chancre)

- 10–90 days after the initial exposure.
- appears at the point of contact, usually the external genitalia.
- The chancre is a firm, painless, relatively avascular, circumscribed, indurated, and superficially ulcerated lesion. termed as 'hard chancre'
- DD from 'soft sore' caused by *Haemophilus ducreyi*.
- The 'hard chancre' of syphilis usually persists for about 4–6 w heals spontaneously.
- painless regional lymphadenopathy develops within 1–2 w after the appearance of the chancre..

Secondary phase

- occurs approximately 1–6 months after the primary infection. This
- ‘flu-like’ syndrome,
- lymphadenopathy
- t symmetrical reddish-pink non-itchy rashes on the trunk and extremities.
- The rash can involve the palms of the hands and the soles of the feet. In moist areas of the body, such as the anus and vagina, the rash often develops into flat, broad, whitish lesions known as condylomata lata.

Secondary phase

- Mucous patches may also appear on the genitals or in the mouth.
- lesions of secondary stage are infectious and harbor active Treponema organisms and therefore patients in this stage are most contagious.
- Other common symptoms of this stage include fever, malaise, sore throat, weight loss,

Latent syphilis

- Following the secondary stage, there oftenoccurs a period of quiescence known as ‘latent syphilis’.
- No clinical manifestations are evident during this phase
- all the lesions of secondary stage have disappeared.
- The diagnosis during this period is possible only by serological tests. In many cases, this phase is followed by natural cure

Tertiary syphilis:

- Manifestations of tertiary syphilis may appear after several years.
- Tertiary syphilis usually occurs 1–10 years after the initial infection and is characterised by the formation of **gummas**, which are soft, tumour-like balls of granulomatous inflammation.
- Other characteristic features of untreated tertiary syphilis include neuropathic joint disease (characterised by degeneration of joint surfaces resulting in loss of proprioception), neurosyphilis, and cardiovascular syphilis.

Investigations

- **Dark-field microscopy**
- ***Non-treponemal tests*** **: Kahn's test, rapid plasma regain (RPR), and venereal disease research laboratory (VDRL)**
- ***Treponemal tests*** **: Microhaemagglutination assay for *T. pallidum* antibodies (MHA-TP), and fluorescent treponemal antibody absorption (FTA-ABS).**

Management

- All women must be offered screening for other STIs including HIV.
- Primary, secondary, and early latent stages of syphilis can
- generally be treated with a single-dose intramuscular (IM) injection of 2.4 million units of benzathine penicillin-G,
- OR a single dose of azithromycin can be administered in a single dosage as the second-line therapy

Management

- **neurosyphilis can be treated with procaine penicillin-G along with concomitant oral probenecid .**
- ***Management in pregnant women*:**
- **In the third trimester of pregnancy,**
- **second dose of benzathine penicillin-G must be administered 1 week after the first dose.**

Complication of Syphilis

- Transplacental passage of treponemal infection can lead to:
- polyhydramnios,
- stillbirths,
- miscarriage,
- preterm labour,
- Hydrops congenital syphilis, etc.

Acquired immuno-deficiency syndrome AIDS

- caused by a retrovirus **human immuno-deficiency virus (HIV)**
- AIDS refers only to the last stage of the HIV infection.
- vulnerable to life- threatening opportunistic infections, neurological disorders, or unusual malignancies
- Fatal, life long infection, pandemic
- Affecting both industrialized and developing countries

Mode Of Transmission

- Sexual transmission
- Blood contact
- Maternal fetal transmission
- I.P is uncertain, (from a few months to 10 years or even more) from HIV infection to the development of AIDS . The virus can lie silent in the body for many years.

Clinical Manifestations

- **The clinical features of HIV infection have been classified into four broad categories**

1. **Initial infection with the virus and development ofantibodies**
2. **Asymptomatic carrier state**
3. **AIDS-related complex (ARC)**
4. **AIDS.**

Syndromic approach to STD

- The traditional method of diagnosing STD is by laboratory tests.
- However, these are often not available or are expensive.
- Since 1990 WHO has recommended syndromic management of STDs in patients presenting with consistently recognized signs and symptoms of STD.

Syndromic approach to STD

- The syndromic approach is a scientifically derived approach and offers accessible and immediate treatment, that is cost effective and efficient, management of STD using flowcharts
- Many different agents cause sexually transmitted diseases. However, some of these agents give rise to similar or overlapping clinical manifestations.

Common syndromes and sequalae of STD

- Male urethritis
- Lower genital syndromes in women
- Vaginitis/cerVicltls/urethritis
- Genital ulceration
- Proctitis/colitis
- Salpingitis
- Epididymitis/orchitis
- Infertility/ectopic pregnancy
- Postnatal and perinatal morbidity
- Hepatitis/hepatic carcinoma
- Genital carcinoma
- Acquired Immuno Deficiency Syndrome (AIDS

Syndromic management of STD

1. Management of urethral discharge in males
2. Management of vaginal discharge
3. Management of lower abdominal pain in female
4. Management of genital ulcer
5. Management of scrotal swelling
6. Management of inguinal bubo

Control of STDs

- Primary prevention focus the prevention of infection.
- Secondary prevention focus minimizing the adverse health effects of infection,
- or usually a combination of the two.

Control of STD

- The control of STD may be considered under the following:
- 1. Initial planning
- 2. Intervention strategies
- 3. Support components
- 4. Monitoring and evaluation

1. Initial planning

Problem Definition

- This is an important first step, since such information is usually inadequate or nonexistent in most cases
- The disease problem must be defined in terms of prevalence, psychosocial consequences and other health effects
- By geographic areas and population groups, with the aid of sero-epidemiological surveys and population surveys.

Establishing Priorities

- Depend upon health problem considerations (e.g., magnitude, consequences)
- Feasibility of control (e.g., availability of adequate resources, social and political commitment).
- Priority groups may be categorized on the basis of age, sex, place of residence, occupation, drug addiction, etc

Setting Objectives

- Priorities must be converted into discrete, achievable and measurable objectives.
- Reduce the magnitude of the problem, in a given population and a stated time.
- Objectives should be unambiguous and quantifiable.
- Broad coverage of the population is crucial for effective STD control.

Considering Strategies

- A variety of intervention strategies should be implemented
- Planners must define the mixture of strategies that appears to be most appropriate to the setting.
- Refer to planning cycle

2. Intervention Strategies

Case detection

- Case detection is an essential part of any control programme.
- The usual methods of early detection in a STD control programme are :

1. Screening
2. Contact tracing
3. Cluster teasting

Screening

- The testing of apparently healthy volunteers from the general population for the early detection of disease .
- High priority is given to screening of special groups, e.g . pregnant women, blood donors, industrial workers, army, police, refugees, prostitutes, restaurant and hotel staff etc.
- Availability of an appropriate test is critical .
- sensitivity, specificity, and predictive value of a test are imp

Contact Tracing

- Term used for the technique by which the sexual partners of diagnosed patients are identified, located, investigated, and treated .
- This is one of the best methods of controlling the spread of infection.
- Patients are interviewed for their sexual contacts by specially trained staff.

Contact Tracing

- The key to success in contact tracing is the patient himself who must disclose all sex contacts **voluntarily**.
- In some parts of USA, contacts are sought as quickly as possible using telephone, telegram and other rapid means of communication.
- The contacts are then persuaded to attend a STD clinic for examination and treatment

Cluster Testing

- Patients are asked to name other persons of either sex who move in the same socio- sexual environment.
- These persons are then screened (e.g., blood testing).
- This technique has been shown almost to double the number of cases found

Case Holding and Treatment

- Adequate treatment of patients and their contacts is the mainstay of STD control.
- Patients suffering from STDs tend to disappear or drop out before treatment is complete.
- Effort should be made to ensure complete and adequate treatment.
- WHO drew up a list of recommendation for TTT STDs recently

Epidemiological Treatment

- Epidemiological treatment or contact treatment is the keystone of the control campaigns .
- Administration of full therapeutic dose of treatment to persons recently exposed to STD while awaiting the results of laboratory tests .
- Should be combined with a venereological examination and the tracing of contacts revealed by that examination.

Personal Prophylaxis

- Mechanical contraceptive barriers (e.g., condoms and the diaphragms)
- Barrier methods+ spermicides.
- Exposed parts should be washed with soap and water as soon after contact.
- Vaccines : The development of a vaccine for hepatitis B has raised hopes that vaccines will be found for other STDs.

Health Education

- Health education is an integral part of STD control programmes.
- The principal aim is

1. help individuals alter their behaviour in an effort to avoid STDs
2. minimize disease acquisition and transmission.

- The target groups may include:

1. the general public
2. patients
3. priority groups &community leaders, etc..

Support Components

1. STD clinic

- The starting point for the control of STDs
- Consultations, investigations ,treatment , contact tracing and all other relevant services are available.
- Ideal services are free ,easly accessible to patient, available for long hours each day.
- Treating female patients separately .
- Mantian anonymity

2. Laboratory Services

- Adequate laboratory facilities and trained staff are essential .
- It provides a basis for:
- correct etiological diagnosis & treatment decisions,
- contact tracing;
- surveillance of morbidity
- detection of antimicrobial resistance.

3. Primary Health Care

- Current trend is to integrate STD control activities into PHC.
- To provide effective treatment to the greatest number of cases in the community.
- Based on the principles of universal coverage, community participation, equity and intersectoral coordination is ideally suited to control STD in the community

4. Information System

- The basis of an effective control programme of any CD.
- It is a prerequisite for effective planning, coordination, M&E.
- Three types of data requirement :

1. clinical notification
2. laboratory notification, and
3. Surveillance.

5. Legislation

قال تعالى "ولا تكرهوا فتياتكم على البغاء ان اردنا تحصنا لتبتغوا عرض الحياة الدنيا "

قال رسول الله"تركت فيكم ما أن تمسكتم به لن تظلوا بعدي أبدا"

- Many countries are still far away in enacting suitable legislation for the control of STDs .
- The purpose of legislation to encourage patients to seek early treatment and name their sexual contacts, to screen high risk groups, to improve notification by GP, health education , etc.

Social Welfare Measures

- STDs are social problems with medical aspects.
- "social therapy" which would prevent or control the conditions leading to promiscuity and STDs.

Social measures to control STD

1. rehabilitation of prostitutes
2. provision of recreation facilities in the community
3. provision of decent living conditions;
4. marriage counselling
5. prohibiting the sale of sexually stimulating literature, pornographic books and photographs, control internet

غزو ثقافي

Monitoring and Evaluation

- Critical aspect of effective management.
- Show if the activities have been performed in a satisfactory way.
- Provides a more direct measure of programme **effectiveness**
- Used to determine the **appropriateness** of the selected intervention strategies for a particular setting.

National STD control programme

Future ????

THANK YOU

Reproductive Health

Dr Kawkab

Objectives

- Define of Reproductive Health (RH
- RH Right
- RH Components & RH service
- Reproductive Health worldwide & Yemen
- Emergency Obstetric and New born care

Definition of Health

"A state of complete physical, mental and social well-being and not merely the absence of disease or infirmity."

Reproductive Health Definition

"A state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity, regarding reproductive system"

Definition of RH

Reproductive health addresses

- **Reproductive processes**
- **Functions and system**
- **All stages of life**

Definition of RH

- **Reproductive health, therefore, implies that people are able to have:**
- **A responsible, satisfying and safe sex life**
- **Have the capability to reproduce**
- **Freedom to decide if, when and how often to do so. FP**

Definition of RH

- The right of men and women to be informed about the method and health services & to have access to
- Safe
- Effective
- affordable(cost effective) and
- acceptable methods

RH Target Group

- ✓ **Men &women in reproductive age**

To raise their level of awareness regarding RH

RH Target Group

- ✓ **Youth& adolescent :**
  - **Decreased risk of exposure to STI&STD.**
  - **To carry their health and family responsibilities later on.**

RH Target Group

- ✓ **Menopause (women after age of reproduction)**

For prevention and early detection of gynecological tumors and disease

- ✓ **Neonate &child**

Reproductive Health Right

- The right of men and women to be informed about the method and health services
- To have access to safe, effective, affordable(cost effective) and acceptable methods
- Right of access to appropriate health care services enable women to go safely through pregnancy and childbirth and provide couples with the best chance of having a healthy infant.

Reproductive Health Right

1. Right on the high quality reproductive health services through primary health care prevent mother exposure to any health risk or hazards "Maternal health care
2. Right on family planning
3. Right of offering child and mother health care

Reproductive Health Right

4. Disseminating information and health education for women

5. Encouraging man on the responsibility of reproductive attitude.

6. Improvement of RH services including FP and avoidance of abortion and it's complication

Islam Reproductive Health Right

- Right on well upbringing
- Right on partner choice
- Right on infancy
- Right on work
- Right on inheritance
- Right on opinion
- Right on own
- Right healthy reproduction
- Right on education

Reproductive Health Care Defintion

constellation of methods, techniques and services that contribute to reproductive health and well-being by preventing and solving reproductive health problems.

Components of Reproductive Health program

- Safe motherhood (ANC, PNC, safe delivery).
- Counseling &FP.
- Control and treatment STD
- Treatment Infertility
- Adolescent &youth care
- Menopause care
- Early detection of women cancers

Reproductive Health Strategy

1. Safe mother hood initiatives 1987
2. Millennium Developmental goals 2000
3. Global strategy women and child health 2009.
4. National reproductive health strategy (NRHS) 2011-2015
5. NRHS 2017-2021
6. SDG 2030

Indicators Of Reproductive Healthcare Services

- **The total fertility rate**
- **The availability of contraceptive measures& use of contraception**
- **Skilled health staff attends to birth**
- **The percentage of pregnant women who receive antenatal**
- **Maternal mortality rates**

Diseases Affecting Reproductive Health

- **Pelvic Inflammatory Diseases**
- **Ectopic pregnancy**
- **Abortion**
- **Anemia & iron deficiency**
- **STD**
- **Fistula(vesicovaginal & rectovaginal)**

Diseases Affecting Reproductive Health

- **Neuro-psychologicaland mental disturbances**
- **Cervical intraepethelial neoplasia , cervical cancer**
- **Unexplained vaginal bleeding**
- **Breast cancer**
- **CVD with pregnancy**
- **Dysmenorrhea**

The importance of RH

Maternal Mortality

- **Rates of maternal mortality are 19 times higher in developing countries than in developed countries .**
- **children in developing countries are 8 times more likely to die before they reach five years of age . Despite substantial progress on maternal and child mortality neither MDG 4 nor MDG 5 targets were met.**

Maternal Mortality

- **But still every day 808 women die from pregnancy or childbirth related causes.**
- **According to the UNFPA 2017 report this is equivalent to about one woman every two minutes and for every woman dies**
- **Most of these death are preventable.**

Maternal Mortality in Yemen

- **maternal mortality ratio 365 / 100000 live birth,164 /100000 live birth DHS**
- **42% of women death in reproductive age due to pregnancy and delivery complication.(old data)**
- **Neonatal death 37/1000 live birth.**
- **70% of neonatal death can be prevented**

- **Maternal mortality globally & in Yemen ??????????????????assignment**

Maternal Mortality Defintion

- **According to WHO, a maternal death is defined as**
- **” the death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of duration and site of pregnancy, from any cause related to or aggravated by the pregnancy or its management but not from accidental or incidental causes.”**

Late Maternal Mortality

- **Death of a women from direct or indirect obstetric causes more than 42 days but less than one year after termination of pregnancy**
- **15% of complication occurred in normal pregnancy(unexpected ,non preventable).**

Causes of Maternal Mortality

- **Direct obstetric cause: deaths are those resulting from**
- **obstetric complications of pregnant state(pregnancy , delivery, and postpartum)**
- **Intervention**
- **Omission**
- **Incorrect treatment**
- **Chain of events resulting from any of the above.**

Causes of Maternal Mortality

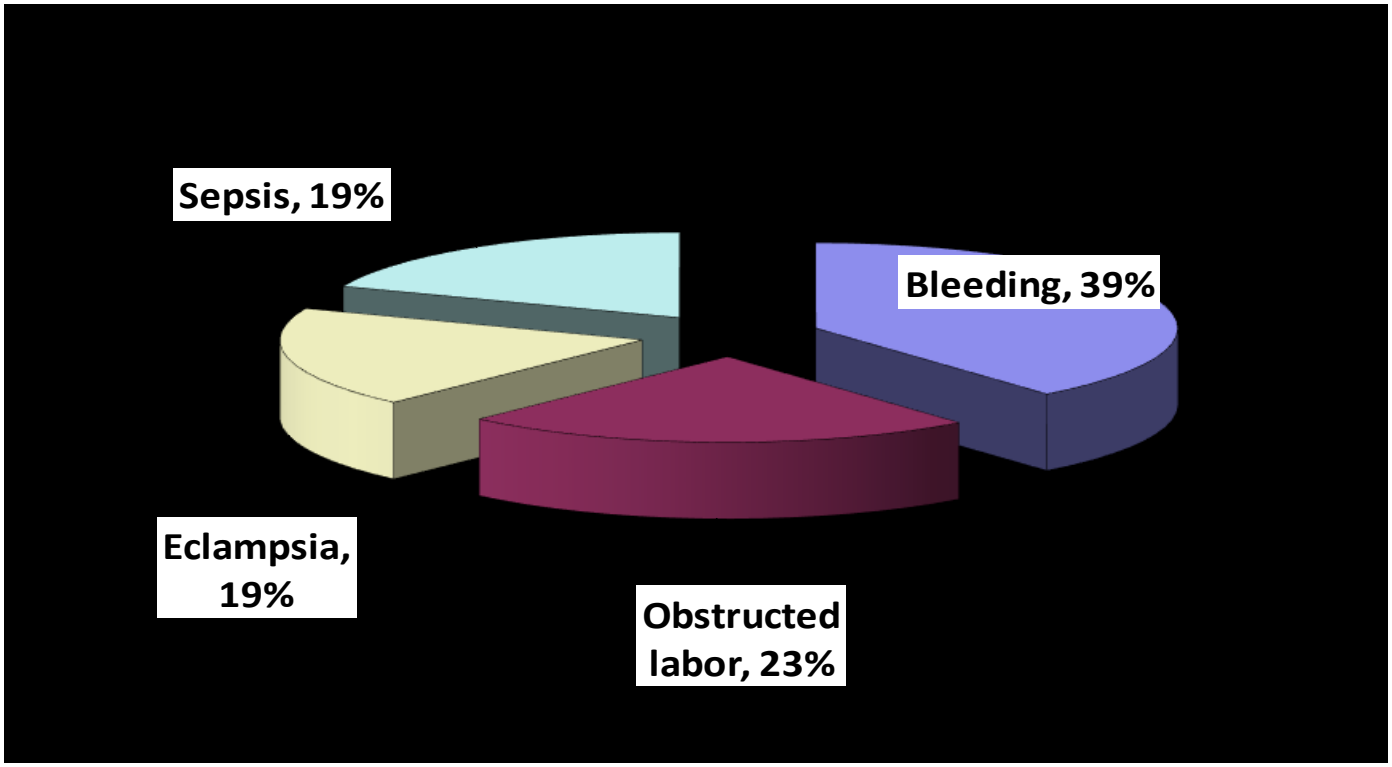
- **Direct causes : deaths due to, for example, hemorrhage, pre-eclampsia/eclampsia or those due to complications of anesthesia or caesarean section are classified as direct obstetric deaths.**
- **Indirect cause: deaths for which thier was a preexixting diseases that was aggrevated by pregnancy.**

Causes of Maternal Mortality

- **Direct causes : deaths due to, for example, hemorrhage, pre-eclampsia/eclampsia or those due to complications of anesthesia or caesarean section**

- **Indirect cause: their was a preexisting diseases that was aggrevated by pregnancy and causing death .**

Direct Causes of MM in Yemen



Direct causes: 80% of maternal Mortality

Indirect Causes of MM in Yemen

- **Malaria**
- **Malnutriton**
- **Anemia**
- **Social &economic factors**
- **Early marriage, early delivery**
- **Illiteracy & poverty**
- **Malpractice and cultural beliefs**

Three delays

1. **Delay in deciding to seek care**
2. **Delay in reaching care in time**
3. **Delay in receiving adequate treatment**

Statistical Measurement of MM

Maternal mortality ratio:

Number of maternal deaths during a given time period per 100 000 live births during the same time-period.

Maternal mortality rate:

Number of maternal deaths in a given period per 100 000 women of reproductive age during the same time-period.

Challenges in Developing Countries to RH

- **poor coverage**
- **Poor quality of health care services**
- **Inadequate water and sanitation**
- **poor infrastructure**

Challenges in Developing Countries to RH

- **Low food security**
- **Limited education**
- **Limted economic opportunity.**

Challenges of RH in Yemen

- lack of awareness
- poor resource availability
- shortage of water food energy
- war
- Destruction of health facilities
- Fragile health system
- COVID 19

Essential Obstetric Care

Provide the basic maternity services to all pregnant women

Essential Obstetric Care

- **Early registration of pregnancy (within 12-16 Wk).**
- **Provision of minimum 3/4 antenatal check ups to detect any risk/complication so that appropriate care including referral could be taken in time.**

❖ **ANC update of WHO more than 8 visits**

Assignment ??

Essential Obstetric Care

- **Provision of safe delivery at home or in an institution**
- **Provision of three postnatal check ups to monitor the postnatal recovery and to detect complications**

Emergency obstetric care

- **Complications associated with pregnancy are not always predictable**
- **Emergency obstetric care is an important intervention to prevent maternal morbidity and mortality.**
- **Most effective intervention for managing maternal complication and improving maternal survival.**

Emergency obstetric care

- **Supply of emergency obstetric kit**
- **Equipment kit and**
- **Provision of skilled manpower .**
- **Training Traditional Birth Attendants**

Why?? •

Emergency Obstetric and Nerw born Care EmONC

To asses level of care these function are classified into:

1. **Basic Emergency Obstetric and New born care BEmONC**
2. **Comprehensive Emergency Obstetric and New born care CEmONC**

Basic Emergency Obstetric and New born care BEmONCC

Life saving treatments and procedure including:

1. **preantral antibiotics anticonvulsants &utertonics**
2. **Manual removal of the placenta**
3. **Removal of retained products**
4. **Newborn recusations**
5. **Assisted vaginal delivery**

Comperhensive Emergency Obstetric and New born care CEmONCC

BEmONC functions

+

Caesarean sections

+

Blood transfusion

Indicators of RH

1. Maternal mortality ratio:

Number of maternal deaths during a given time period

per 100 000 live births during the same time-period.

2. Maternal mortality rate:

Number of maternal deaths in a given period per 100 000 women of reproductive age during the same time-period.

Indicators of RH

3. Skilled attendant at birth:

Percentage of live births attended by skilled health personnel.

4. Antenatal care visits:

Number of women attended at least four times during pregnancy by any provider for reasons related to the pregnancy

Family Planning

Dr kawkab Al-Haddad

Objective of the Lecture

1. Define FP and their objectives
2. Understand different fertility regulated methods
3. Identify the failure rate of each method
4. Recognize human rights principles guide family planning services

Definition

According to WHO:

Family planning allows individuals and couples to anticipate and attain their desired number of children &the spacing and timing of their births.

It is achieved through use of contraceptive methods and the treatment of involuntary infertility

Definition

- تنظيم الأسرة هو قيام الزوجين بالترا ضي بينهما وبدون اكراة باستخدام وسيلة مشروعة ومأمونة لتأجيل الحمل أو تعجيلة بما يناسب ظروفهما الصحية والاجتماعية والاقتصادية وذلك في نطاق المسؤولية نحو أولادهما وأنفسهما (مؤتمر القاهرة)

Definition

The way of thinking and living that is adopted voluntarily up on the basis of knowledge, attitude and responsible decision of individual and couples in order to promote health and welfare of the family group and thus contribute effectively to social development of the country

Family limitation

- **Limitation the number of children e.g in china**

Against our religion

Objectives

1. To avoid unwanted births
2. To bring about wanted births
3. To regulate interval between pregnancies
4. To control the time at which birth take place in relation to the age of parents
5. To determine the number of children in the family

Goal of family planning is to enable couples and individuals to freely choose how many children to have and when to have them

Aim

- **Aim of FP services is to improve quality of life of people**
- **FP is basic human right**
- **All couples must have the right to decide freely the number of children and spacing between them**

family planning target group

- **Eligible couples : Currently married couple when the female in reproductive age(15-45yr).**
- **Adolescents will be targeted when they are in the reproductive age**
- **Adolescence: the period of life between 10-19 years**

HOW?

Healthcare attendant provides couples with a full range of safe and effective contraceptive methods and gives them sufficient information

To

ensure that they are able to make informed choices.

The present approach in family planning programs is to provide

a cafeteria choice

Offer all methods from which an individual can choose according to his needs and wishes

To

promote family planning as away of life.)

Fertility regulated methods

The methods include all temporary and permanent measures to prevent pregnancy resulting from coitus.

Contraceptive general strategies

(1) prevention of ovulation,

(2) prevention of fertilisation

(3) prevention of implantation

Most effective
Less than 1 pregnancy per 100 women in 1 year

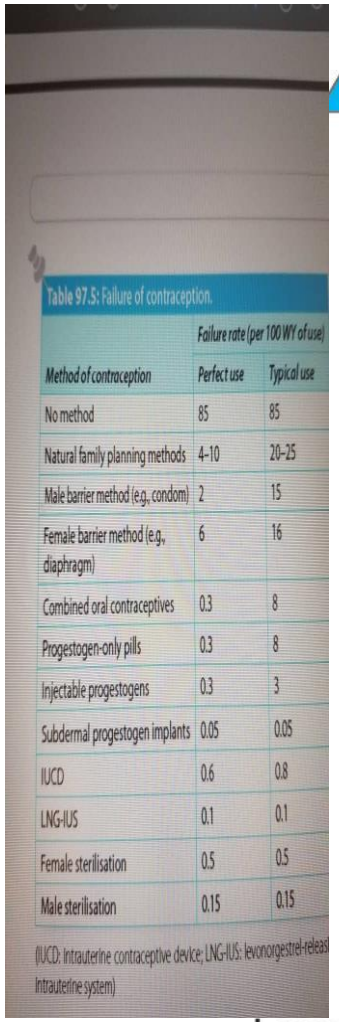


Table 97.5: Failure of contraception.

Method of contraception	Failure rate (per 100 WY of use)	
	Perfect use	Typical use
No method	85	85
Natural family planning methods	4–10	20–25
Male barrier method (e.g., condom)	2	15
Female barrier method (e.g., diaphragm)	6	16
Combined oral contraceptives	0.3	8
Progestogen-only pills	0.3	8
Injectable progestogens	0.3	3
Subdermal progestogen implants	0.05	0.05
IUCD	0.6	0.8
LNG-IUS	0.1	0.1
Female sterilisation	0.5	0.5
Male sterilisation	0.15	0.15

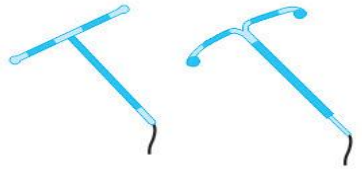
(IUCD: Intrauterine contraceptive device; LNG-IUS: levonorgestrel-releasing intrauterine system)

Least effective
18 or more pregnancies per 100 women in 1 year

Implant

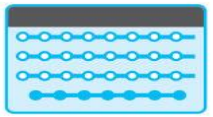
Vasectomy

Female sterilisation



IUC

Injectable



Pills

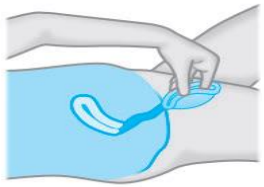
Patch

Ring

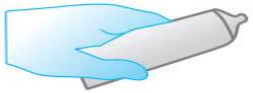
Male condom

Female condom

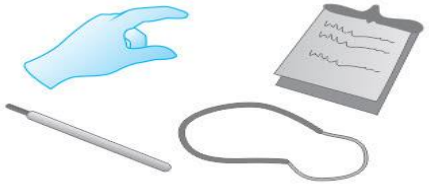
Sponge



Withdrawal



Spermicides



Fertility awareness-based methods

Natural family planning

Vaginal ring

Female condoms

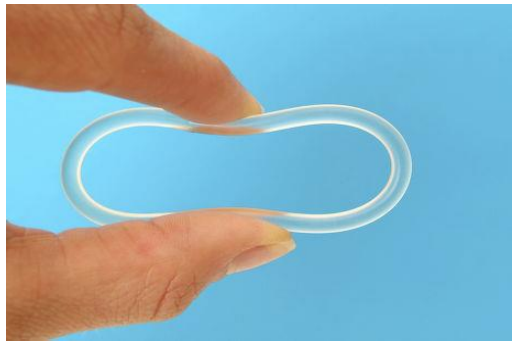
Male condoms

Contraceptive patch

Injectables

Oral contraceptives

Intrauterine devices



Long-acting Reversible Contraception

1. **Non-hormonal methods**

Copper Intrauterine Device (IUD)

2. Hormonal methods

a) **levonorgestrel-releasing Intrauterine system(LNG-IUS)**

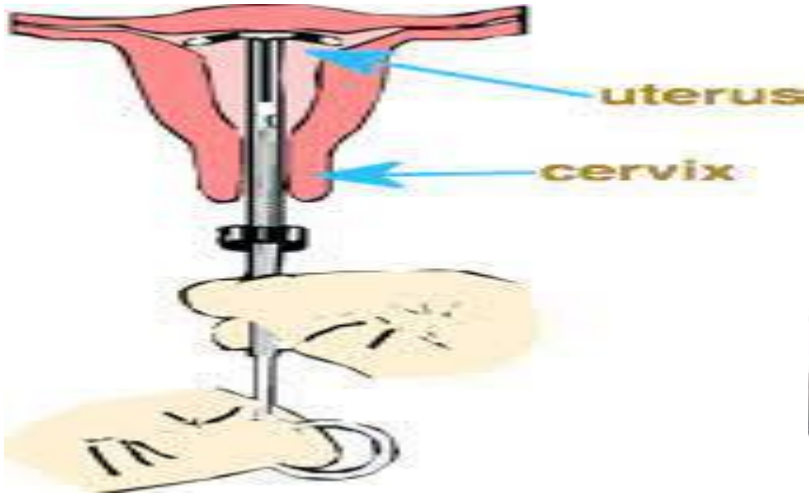
b) **progestogen-only injectables**

c) **progestogen-only implants).**

Intra uterine device

I. Non-medicated
first generation

II. Medicated
-second generation
-third generation



Side effects

- Heavy bleeding and pain
- Pelvic inflammatory diseases(PID)
- Ectopic pregnancy
- May come out accidently if not properly inserted
- perforation

How Effective?

- **One of the most effective and long-lasting methods**
- **Less than 1 pregnancy per 100 women using an IUD over the first year**

Perfectly 6 per 1,000 women who use the IUD

commonly used 8 per 1,000 women .

This means that 992 to 994 of every 1,000 women using IUDs will not become pregnant.

Contraindication

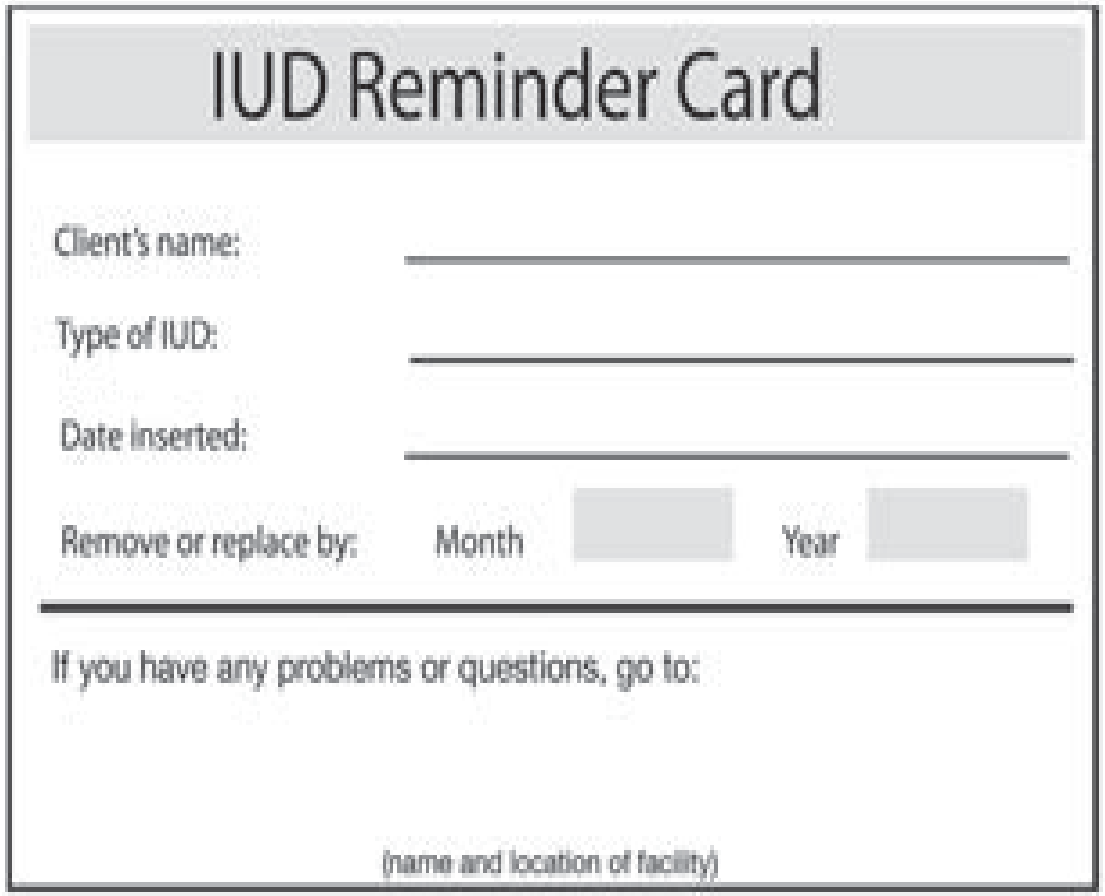
ABSOLUTE:

- Suspect pregnancy
- PID
- Undiagnosed vaginal bleeding
- Pelvic tumours
- Previuos ectopic pregnancy

RELATIVE:

- Anemia
- History of PID since last pregnancy
- Purulant cervical discharge
- Congenital utrine malformation

IUD



IUD Reminder Card

Client's name: _____

Type of IUD: _____

Date inserted: _____

Remove or replace by: Month _____ Year _____

If you have any problems or questions, go to:

(name and location of facility)

- **A follow-up visit after her first monthly bleeding**

or

- **3 to 6 weeks after IUD insertion is recommended.**

Hormonal contraceptive

A. Oral pills

1.Combined pill

2.Progestoge only pill

3.Post coital pill(emergency)

B. Depot (slow release)

1. **Injectables**
2. **Subcutaneous implants**
3. **Vaginal ring**

Combined pill

- Is oestrogen and progestogen
- Is given orally for 21 consecutive days
- Beginning in 5th day of cycle
- Followed by break of 7 days (during which period menstruation occurs),this is considered the first day of the next cycle.
- The effectiveness is affected by certain drug such as rifampicin,phenolbarbital and ampicillin

Combined pill Mechanisms

1. Prevention of ovulation
2. Thickening of mucus at the cervix so sperms cannot pass.
3. Changing the environment of the uterus and fallopian tubes to prevent fertilisation and/or implantation

Patient assessment prior to prescription

1. History

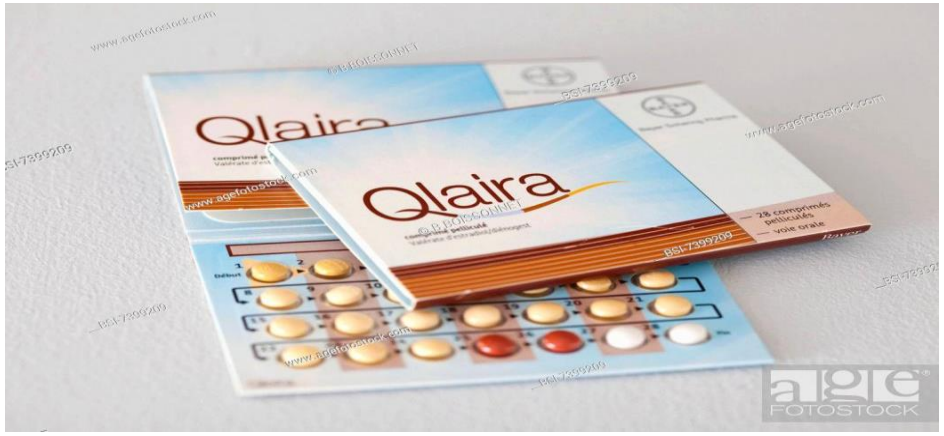
- including potential contraindications, smoking history, and medications.

2. Physical examination should include a blood pressure measurement.

3. Adequate counselling prior to initiation of CHCs may help to improve compliance and adherence

A pelvic examination is not mandatory before prescription.

COMBINED PILLS



Post coital contraception

- **Recommended within 72 hours of unprotective intercourse**
- **Two methods available:**

- **IUD: especially copper device within 5 days.**
- **hormonal : levonorgestrel 0.75 mg tablet and 2nd tablet after 12 hrs.(within 72hrs)**

Combined pill Effectiveness

1. commonly use

7 pregnancies per 100 women using COCs over the first year. This means that 93 of every 100 women using COCs will not become pregnant.

2. Accurate use

<1 pregnancy per 100 women using COCs over the first year (3 per1,000 women).

Combined pills and cancer

- Use of COCs **increase** risk of two cancer

1. **Breast cancer**

some studies find that current users of COCs and women who have used COCs within the past 10 years are more likely to be diagnosed with breast cancer.

2. **Cervical cancer**

Use of COCs for 5 years or more appears to increase slightly the risk of cervical cancer.

Combined pills and cancer

Use of COCs helps protect users from 3 important kinds of cancer

1. **Ovarian cancer**
2. **endometrial cancer. (protection continues for 15 years or more after stopping use of COCs.)**
3. **colorectal cancer.**

Progestogen – only pill(POP)

- It contains only progestogen
- Act by make cervical mucus thick and scanty also inhibit tubal motility and delay transport of the sperm and ovum to uterine cavity

Progestogen only pills

Progestogen only pills effectivness

Breastfeeding women:

- **Commonly used**
- **1 pregnancy per 100 women using POPs over the first year. This means that 99 of every 100 women will not become pregnant.**
- **Perfect (accurate)**

less than 1 pregnancy per 100 women using POPs over the first year(3 per 1,000 women).

Progestogen only pills efffectivness

Less effective for women not breastfeeding:

- **As commonly used**

7 pregnancies per 100 women using POPs over the first year.

This means that 93 of every 100 women will not become pregnant.

- **Perfect use (When pills are taken every day at the same time)**

<1 pregnancy per 100 women using POPs over the first year

Injectable contraceptive

- **DMPA(depot-medroxyprogeserone acetate): intramuscular injection 150mg every 3 months,its action by suppression of ovulation**
- **DMPA-SC 104 mg: contain 104 mg injected sub cutaneous at 3 months interval**
- **NET-EN(norethestrone emanate):every 60 days im injection**

Work primarily by preventing the release of eggs from the ovaries (ovulation).

Injectable (DMPA)

Injectable contraceptive effectiveness

- **As commonly used**

4 pregnancies per 100 women using progestin-only injectables over the first year. This means that 96 of every 100 women using injectables will not become pregnant.

- **When women have injections on time**

less than 1 pregnancy per 100 women using progestin-only injectables over the first year (2 per 1,000 women).

Side effect

- **Disruption of normal menstrual cycle:**
- **Prolonged bleeding**
- **Or become amenorrhoeic**

Subdermal implant

- **Containing 68 mg of etonorgestrel**
- **Long term up to 3 yrs reversible contraception**
- **Release the hormones about 60mcg gradually decrease to 30mcg/ day over 3 yrs**

Subdermal implant insertion

- **Capsule inserted subdermally in inner aspect of the nondominant arm , 6-8 cm above the elbow fold**
- **Inserted between biceps and triceps muscles**
- **Done under local anaesthesia inserted within 5th day of menstrual cycle**

IMPLANON DEVICE

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Subdermal implant follow up

- No routine follow-up is required.
- Review if:
- woman cannot feel the implant
- it appears to have changed shape
- any change to the skin or pain around the site of the implant
- becomes pregnant
- No evidence of a causal association with weight change ??, mood change, loss of libido, or headache.

Subdermal implant effectivness

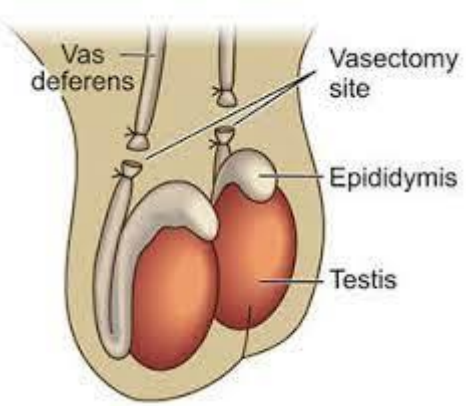
Far less than 1 pregnancy per 100 women using implants over the first year (1 per 1,000 women).

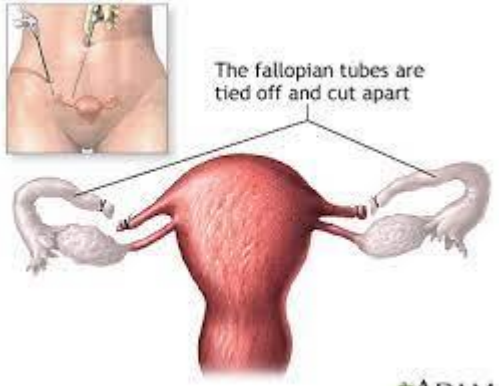
- **This means that 999 of every 1,000 women using implants will not become pregnant. Less than 1 pregnancy per 100 women over the duration of use.**

Natural contraception

1. Abstinence
2. Coitus interruptus
3. Safe period
4. Breast feeding

Terminal methods





Male sterilization :

Vasectomy simple operation can be performed even in primary health centers by trained doctors under local anesthesia .

Female sterilization:

- Minilapratomy& Laparoscopy procedure
- Bilateral tubal ligation
- Resection of both segment of both fallopian tubes
- Two approach abdominally,vaginally

Male sterilization

- Through a puncture or small incision in the scrotum, the provider locates each of the 2 tubes that carries sperm to the penis (vas deferens) and
- cuts or blocks them by cutting and tying them closed or by applying heat or electricity (cautery).
- Semen is ejaculated, but it cannot cause pregnancy

Male sterilization

- 3-month delay in taking effect. The man or couple must use condoms or another contraceptive method for 3 months after the vasectomy.
- Does not affect male sexual performance
- simple .
- faster and less expensive than tubectomy.

Does a woman who has had a sterilization procedure ever have to worry about getting pregnant again?

Sterilization taking decision

Asking questions can help. The provider might ask:

- **"Do you want to have any more children in the future?"**

"If not, do you think your mind could change later?

- **What might change your mind? For example, suppose one of your children died?"**

- **"Suppose you lost your spouse, and you married again?"**

- **“Does your partner want more children in the future?”**

Clients who cannot answer these questions may need encouragement to think further about their decisions about sterilization.

Female sterilization effectivness

- **Less than 1 pregnancy per 100 women over the first year after having the sterilization procedure (5 per 1,000).**
- **This means that 995 of every 1,000 women relying on female sterilization will not become pregnant.**

Male sterilization effectivness

- **Far less than 1 in every 100 will become pregnant in the first year of use of the method. In fact, less than 2 women in every 1,000 will become pregnant.**
- **This means that 998 or 999 of 1,000 women whose partners have had vasectomy will not become pregnant.**

Compare the effectiveness of male and female sterilization?

Complication of tubectomy

- **Ectopic pregnancy**
- **Menstrual irregularities (post PTL syndrome)**
- **Loss of libido**
- **infection**

- **Can sterilization be reversed if the woman decides she wants another child?**
- **Is it better for the woman to have female sterilization or for the man to have a vasectomy?**

Condom

- **Male condoms help protect against STI, including HIV.**
- **Require correct use with every act of sex for greatest effectiveness.**
- **Require both male and female partner's cooperation.**
- **Very few pregnancies or infections occur due to incorrect use, slips, breaks.**

Protection against pregnancy

- **Commonly used, about 13 pregnancies per 100 women whose partners use male condoms over the first year.**

This means that 87 of every 100 women whose partners use male condoms will not become pregnant.

- **Correctly used with every act of sex, about 2 pregnancies per 100 women whose partners use male condoms over the first year.**

Protection against HIV and other STIs

- **Male condoms significantly reduce the risk of becoming infected with HIV when used correctly with every act of vaginal or anal sex.**
- **When used consistently and correctly, condom use prevents 80% to 95% of HIV transmission that would have occurred without condoms**

Method of contraception0.15	Failure Rate	
	Perfect use	Commonly use
No method	85	85
Natural FP	4-10	20-25
Condom (M. barrier method	2	15
Diaphragm(F. barrier method)	6	16
Combined oral contraceptives	0.3	8
Injection progestogen	0.3	3
Implants	0.05	0.05
IUCD	0.6	0.8
LNG-IUS	0.1	0.1
F. sterilization	0.5	0.5
M. sterilization	0.15	0.15

Failure rate of contraception /100WY

Human Rights: Family Planning Providers' Contribution

(http://www.who.int/reproductivehealth/publications/family_planning/human-rights-contraception/en/).

Nine human rights principles guide family planning services

Non-discrimination

Availability of contraceptive information and services

Accessible information and services

Do not ask clients, even young clients, to get someone else's permission to use family planning or a certain family planning method.?????

Religion

Country Regulation

Acceptable information and services

Quality

- **What you can do:**
- **Keep your knowledge and skills up-to-date.**
- **Use good communication skills.**
- **Check that contraceptives you provide are not out-of-date.**

Informed decision-making

Explain family planning methods clearly, including

- **how to use them,**
- **how effective they are, and**
- **what side effects .**
- **Help clients consider what is important to them in a family planning method.**

Confidentiality

- **Do not tell others what your clients have said.**
- **Promptly put away clients' records.**

Privacy

Do not discuss your clients with others except with permission .

When talking with clients, find a place where others cannot hear.

Participation

What you can do:

Ask clients what they think about family planning services.

Act on what they say to improve care.

Accountability

What you can do:

Hold yourself accountable

- **for the care that you give clients and**
- **for their rights.**

Client Rights

- **Information**
- **Access to services**
- **Informed choice**
- **Safe services**
- **Privacy and confidentiality**
- **Dignity comfort and expression of opinion**
- **Continuity of care**

Provider Needs

- **Information ,training, updates**
- **Supplies , equipment , infrastructure**
- **Good Management ,facilitative supervision**

Benefits of family planning and contraception

1. **Economic development**
2. **Maternal and child health,**
3. **Education, and**
4. **Women’s empowerment.**

Preventing maternal morbidity and mortality

1. **Reducing the number of times a woman is exposed to the risks of pregnancy.**
2. **Helping women avoid unintended and closely spaced pregnancies (a study in Bangladesh found that very short pregnancy intervals are linked with 7 times increased risk of induced abortion).**
3. **Helping women avoid more than 4 births or births after 35 years of age .**

Who provides contraception?

- Task Shifting and Task Sharing

Pelvic Inflammatory Disease

Dr samaya

PID divide into

*١ Primary PID

Sexual active women

*٢ Secondary PID

Intrauterine device

DIC

Caesarean section

مابيحصل الحمل اثناء لماذا PID ?

عدوى اي ومابيحصل الجنين باغشية cervical os ال بيغلق لانه

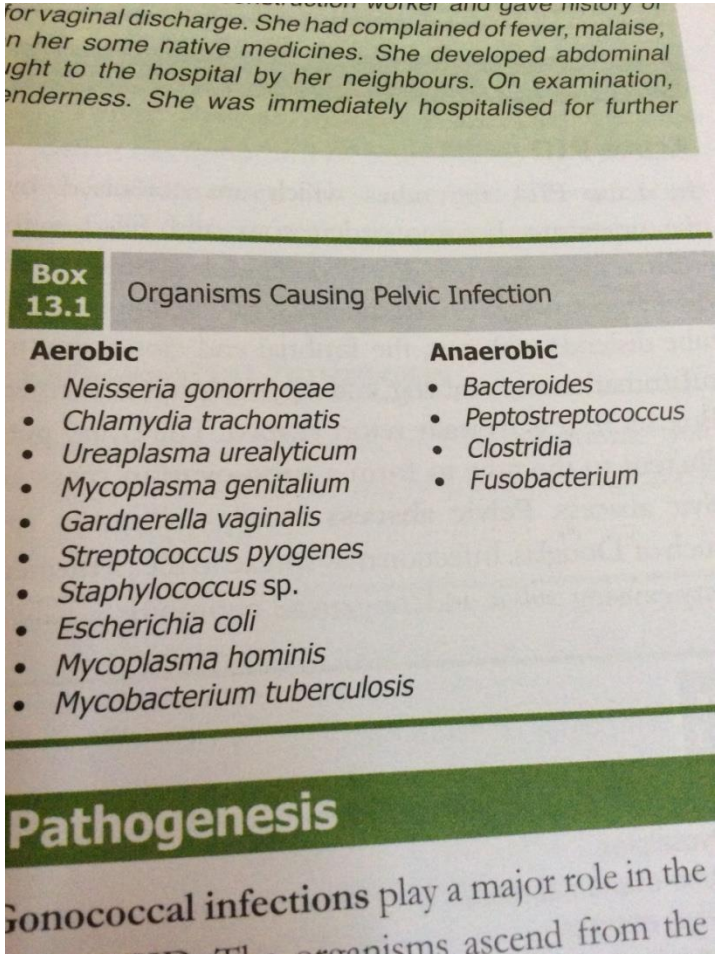
إجهاض المراة مع بيحصل حصل واذا c

Early diagnosis and treatment are believed to be key elements in the prevention of long-term sequelae, such as infertility and ectopic pregnancy

PID is primarily a disease of sexually active women.

The two most important sexually transmitted organisms associated with acute PID are Chlamydia trachomatis and Neisseria gonorrhoeae.

Polymicrobial infection



for vaginal discharge. She had complained of fever, malaise, n her some native medicines. She developed abdominal ight to the hospital by her neighbours. On examination, enderness. She was immediately hospitalised for further

Box 13.1 Organisms Causing Pelvic Infection

Aerobic

- *Neisseria gonorrhoeae*
- *Chlamydia trachomatis*
- *Ureaplasma urealyticum*
- *Mycoplasma genitalium*
- *Gardnerella vaginalis*
- *Streptococcus pyogenes*
- *Staphylococcus* sp.
- *Escherichia coli*
- *Mycoplasma hominis*
- *Mycobacterium tuberculosis*

Anaerobic

- *Bacteroides*
- *Peptostreptococcus*
- *Clostridia*
- *Fusobacterium*

Pathogenesis

Gonococcal infections play a major role in the

ganisms ascend from the

- PID is a polymicrobial infection.

- Acute PID is an ascending infection caused by cervical as well as the vaginal microflora, including anaerobic organisms

- Cervical microorganisms - Chlamydia trachomatis and Neisseria gonorrhoeae)
- Vaginal microflora, including anaerobic organisms, enteric gram-negative rods, streptococci, genital mycoplasmas, and Gardnerella vaginalis, which is associated bacterial vaginosis

Bacterial vaginosis results in complex alterations of the normal vaginal flora, which may alter host defense mechanisms in the cervicovaginal environment

Types

- Acute PID
- Chronic PID

Classification of PID

1_acute PID
Causes
Emergency department

2_Subacute PID

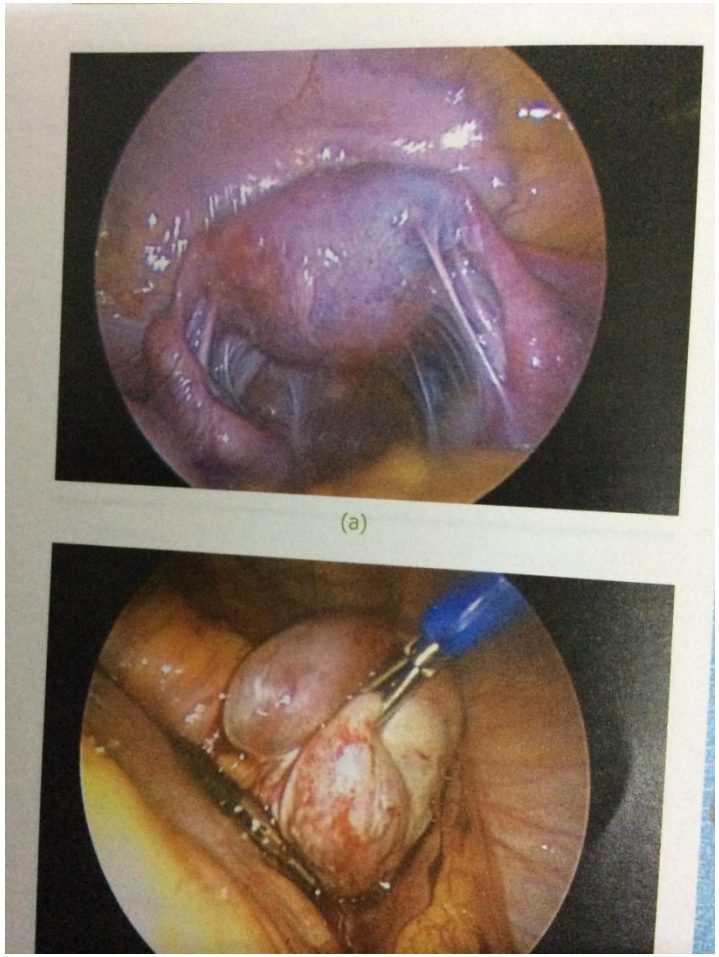
3_ Chronic PID

Causes
TB
Infertility

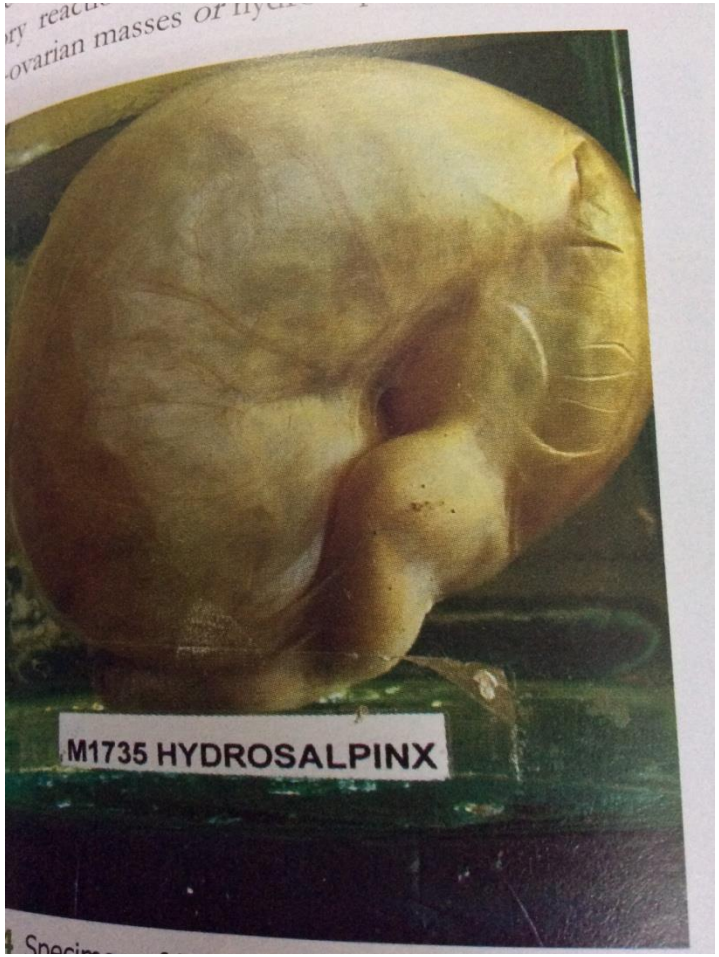
Pathology of Acute PID

- Inflammed, fluid filled tubes
- Pyosalpinx
- Tubo- Ovarian Abscess
- Pelvic abscess
- Pelvic/general peritonitis.

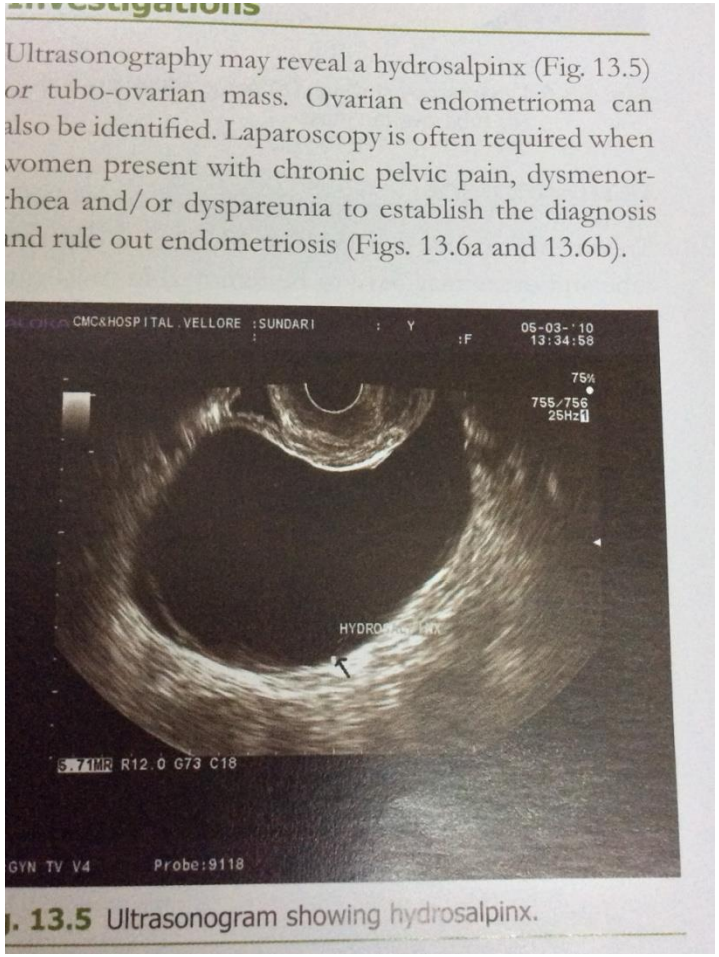
Pelvic adhesions



Hydrosalpinx



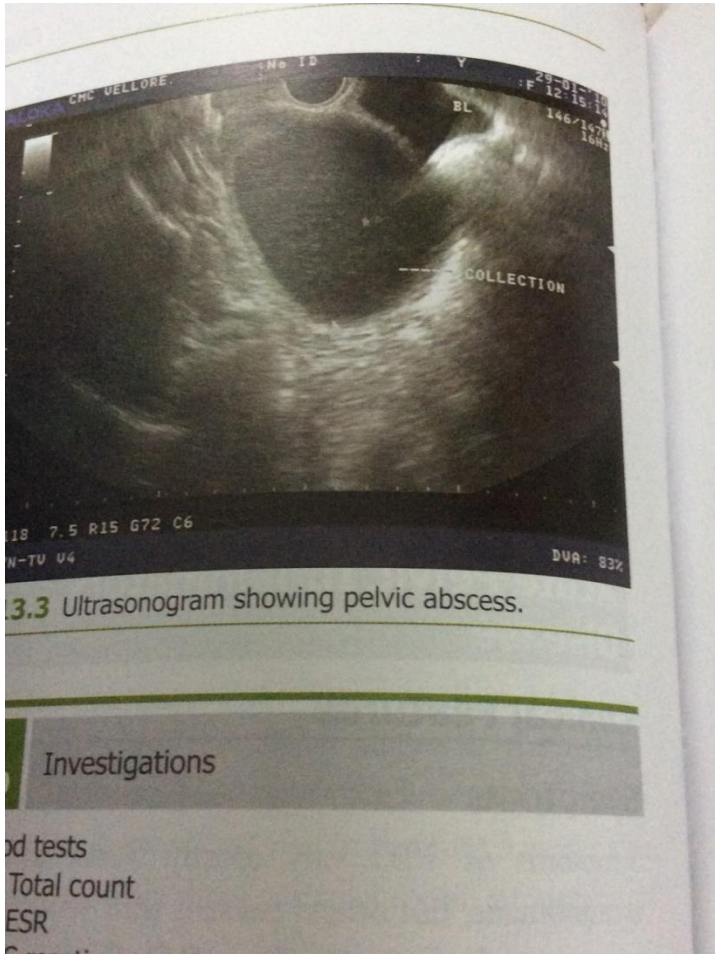
USG showing hydrosalpinx



Ultrasonography may reveal a hydrosalpinx (Fig. 13.5) or tubo-ovarian mass. Ovarian endometrioma can also be identified. Laparoscopy is often required when women present with chronic pelvic pain, dysmenorrhoea and/or dyspareunia to establish the diagnosis and rule out endometriosis (Figs. 13.6a and 13.6b).

13.5 Ultrasonogram showing hydrosalpinx.

Pelvic abscess- usg TVS



Pyosalpinx seen in USG

asonogram revealing pyosalpi

Tubovarian mass in USG

118 7.5 R17 G72 C6
1 Ultrasonogram revealing pyosalpinx. It is elongated and filled with echogenic pus.
200/2011
19Hz
ovary
R08 G72 C6
Ultrasonogram showing tubo-ovarian mass. The
ovary (arrow) is seen adherent to the

- Fitz Hugh Curtis Syndrome – Inflammation of the liver capsule can occur with chlamydia and gonococcal infection.

- Patient has right upper quadrant pain & liver tenderness.

Fitz Hugh Curtis Syndrome – Inflammation of the liver capsule can occur with chlamydia and gonococcal infection.
Patient has right upper quadrant pain & liver tenderness.
Also called "prehepatic adhesion "
For diagnosis do laparoscopy
To see "violin string" adhesions of parietal peritoneum to liver.
Acute emergency
هذا المريض يحتاج hospitalization
aantibiotics
supportive

Chronic PID

Women who are inadequately treated, untreated or have recurrent infections, chronic PID ensues.

Chronic PID- Pathology

- Tubo ovarian mass
- Pyosalpinx pus

- Hydrosalpinx
- Frozen pelvis

Risk Factors for PID

- Young age
- Low socioeconomic status
- Multiple sex partners
- Unmarried/ widowed women
- Past h/o STI
- Vaginal douching
- IUCD for 3 weeks after insertion
- Smoking/ substance abuse.

Pathogenesis

1*Decrease astrogen hormone lead to ascending of bacteria____Cervical thin mucus called astrogen domnant cervical thin mucus

2* thin mucus ___asending sperms ____cause infection

3*intrauterine insertion

4*viginal Douche

5*Intercorse during menstrual cycle

6* Intercorse after Caesarean Section____lead to sepsis

7*Human immunity

Chlamydia trachomatis	Neisseria gonorrhoeae
_attachement colmnar cells in endometrum, ovary and fallopin tube reaction with immune cells _exudate _Sometimes granuloma Chylamydia body مواصفات المرأة الذي عندهاCalmadia body By piobsy mucopurulent cervicitis Treatment Two-weeks recurrent antibiotics suppositories	attachment to secretary cells of uterus ___Endometritis Fallopin tube block destrection in immune cells and cillia in the fallopin tube reaction____ _fluid _exudate _ secendary infection Causes Pyosalpinx hydrosalphax Tubo ovarian absess Female useless

Complications/ Sequelae

- Pelvic peritonitis
- General peritonitis
- Rupture of tubo- ovarian abscess
- Sub diagphragmatic/ perinephric abscess
- Septic thrombophlebitis
- Septiceamia

Sequelae

- Ectopic pregnancy
- Infertility
- Chronic pelvic pain

Clinical picture of PID

Non tuberculosis salpengitis	Tuberculosis salpengitis
Classical symptoms Mucopurulent cervicitis discharge اهم عرض Midline abdominal pain Vaginal bleeding Sometimes nuasea vomiting Urethritis dysuria Preappendicitis Perihepatitis	Past history ٣٨% Contact with TB patients Symptoms Dysemonria Abnormal vaginal bleeding Infertility لوعملنا الفحص يكون عندها tuberculosis granuloma في قناة فالوب يسبب TBعندما يكون ال Narrow Bleeding Fimbriae closed Bilateral

Acute PID - Symptoms

- Lower abdominal pain
- Abnormal vaginal discharge
- Fever
- AUB
- Dyspareunia- deep
- Nausea, vomiting,diarrhea, tenesmus
- Right upper quadrant pain

Acute PID - signs

- Lower abdominal tenderness
- Liver tenderness
- Signs of peritonitis
- Abnormal vaginal discharge
- Mucopus exuding from os
- Cervical motion tenderness
- Adenexal tenderness/ mass
- Fullness in POD- pelvic abscess

Diagnosis

History

CBC

vaginal s wab____mucoplurent with discharge

Ultrasound__ overy, uterus ,liver

Laparoscope in acute case

odema in the tube

enlarge

Exudate

Erythema

adrenal varicose

Histopathology

Endometrial piobsy

Culture

Investigation – acute PID

- Hgm including TLC & DLC, ESR, CRP
- Endocervical d/s for pus cells & NAAT for chlamydia & gonorrhoea
- Vaginal d/s for wet saline & KOH test
- Urine c&s
- TVS- TOmass, Pyo or hydrosalpinx,Pelvic abscess, free peritoneal fluid & to exclude ectopic preg

D/D of acute PID

- Ectopic pregnancy
- Torsion/ rupture of ovarian cyst
- Endometriosis
- Acute appendicitis
- UTI
- Diverticulitis
- IBS
- IBD

- Laparoscopy is considered the gold standard for diagnosis.
- But not performed as routine
- Indicated in patients who do not respond to initial therapy or diagnosis is doubtful.

Management of acute PID

- Assess the need for hospitalization
- Antimicrobial therapy
- Treatment of partners
- Counselling
- Assessment of response to therapy
- Decision regarding surgical intervention
- Follow-up for sequelae.

CDC guideline for acute PID –inpatient therapy

Regimen A

Cefoxitin (2 g intravenously every 6 hours) or cefotetan (2 g IV every 12 hours)

plus

Doxycycline (100 mg orally every 12 hours).

I/V therapy is discontinued 24 hrs after patient is fever free..

Continue oral doxy for 14 days.

Regimen B

Clindamycin (900 mg intravenously every 8 hours) plus gentamicin loading dose (2 mg/kg of body weight) followed by a maintenance dose (1.5 mg/kg) every 8 hours. Single daily intravenous dosing of gentamicin may be substituted for three times daily dosing

Note

When we use Gentamycin

Renal function test must be NORMAL

Indications for surgical intervention

- USG guided pus aspiration- Pelvic abscess, subhepatic abscess
- Posterior colpotomy- Pelvic abscess
- Laparoscopic aspiration of pus or drainage or adhesiolysis
- Laparotmy for to abscess/ or rupture of same or multiple collection in abdomen
- Salpingoopherectomy.

Management of Sexual partners

- Contact partners within 6 months of onset of disease.
- Screen for gonococcal/chlamydial infection
- If screening not possible, start empirical therapy.
- Avoid intercourse till the partner completes treatment.

Counseling

- Early treatment reduces the risk of sequelae but does not eliminate it.
- Barrier contraception reduces risk
- Recurrence of infection increases the risk of infertility.
- Sexual partner must be treated.

Chronic PID- symptoms

- History of previous infection.
- Lower abdominal pain
- Deep dyspareunia
- Congestive dysmenorrhoea
- Menorrhagia, polymenorrhoea, polymenorrhagia
- Chronic pelvic pain
- Infertility

Signs of Chronic PID

- Abdominal examination- Tenderness, mass arising from pelvis
- Per speculum examination- Vaginal/ Cervical discharge
- Pelvic examination- Fixed r/v tender uterus, adenexal tenderness, pelvic mass, hydrosalpinx, tubo- ovarian mass.
- Frozen pelvis

Differential diagnosis

- Endometriosis
- Chronic ectopic pregnancy

Differential diagnosis also
Cholecystitis
Polynephritis
tubo ovarian cyst
له ونعمل أسبوع لمدة الحالة ننوم مؤكد غير لوالتشخيص
للتشخيص نوصل نحاول أجل من investigation

the need for hospitalization in PID
في حالة
1. pelvic abscess
2. adolescent women
3. Sever pain
4.Fitz-Hugh–Curtis syndrome
5. Patients that don't follow up
مثل النساء اللواتي يأتين من مناطق بعيده كالقرى.

Investigation

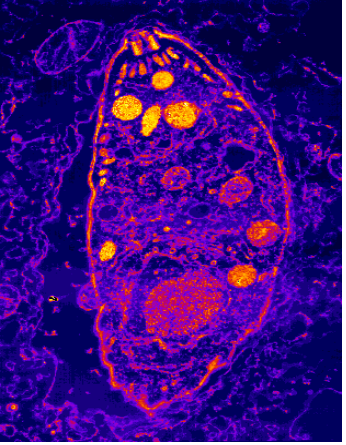
- USG may reveal hydro/ pyosalpinx, TO mass, Ovarian endometrioma
- Laparoscopy may be required for diagnosis & therapy.

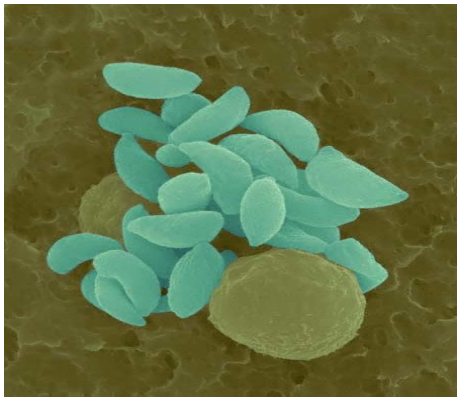
Management of Chronic PID

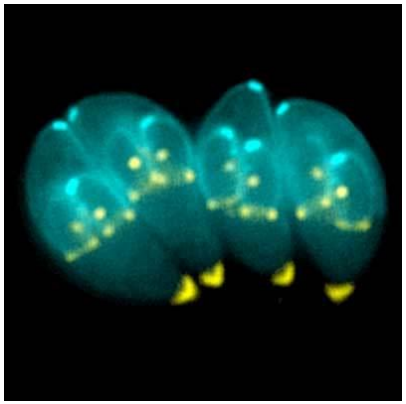
- Laparoscopy- adhesiolysis, salpingo-opherectomy
- Laparotomy- Adhesiolysis, Salpingo-opherectomy.
- Hysterctomy with bilateral salpingo-opherectomy

Fitz hugh curtis syndrome

هذا المرض عن ونقرا مهم







Toxoplasmosis

Is a protozoan shed in cats that can be spread to humans by a variety of way s.

Presented by

What is Toxo?

A fetus may contract toxoplasmosis through the placental connection with its infected mother

The mother may be infected by:

Improper handling of cat litter

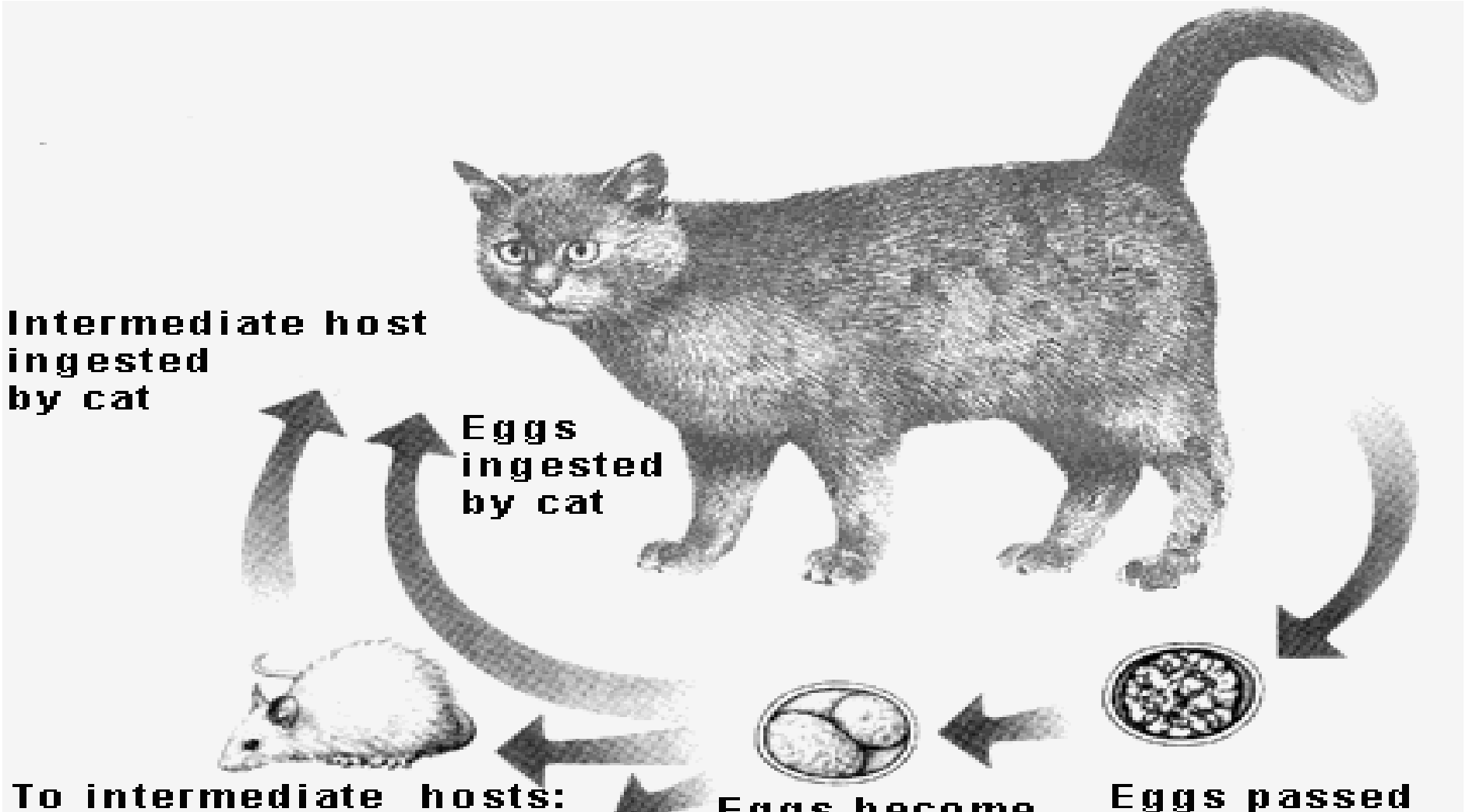
Handling or ingesting contaminated meat

A.D.A.M.

Transmission

- Eating infected meat: Primary source of transm ission to humans
- Ingestion of recently infected feces.
- Transmission of mother to fetus (it is zoonotic)
- 2 infection stages
    - Latent: bradyzoites in nervous tissue
    - Acute: flu like symptoms
        - swollen lymph nodes (neck, axillae, groin)
        - Pain lasting month or longer

Lifecycle of Toxoplasmosis



Who is at risk?

- Most species of animals and birds can contract Toxo. Although cats are the definitive host of the parasite.
- People who are most likely to develop severe toxoplasmosis include infants born to mothers who are newly infected with Toxoplasma gondii during or just before pregnancy.
- Persons with severely weakened immune systems, such as individuals with HIV/AIDS.
- Those taking certain types of chemotherapy.
- Those who have recently received an organ transplant.

Clinical signs

- Most primary infections produce no symptoms.
- Time between exposure to the parasite and symptom development is 1-2 weeks.
- Mostly in severely immunocompromised or very you ng animal.
- Cats: Lung and eye; In muscles : protozoal myositis
- Dogs: GI, neurologic and respiratory

Symptoms

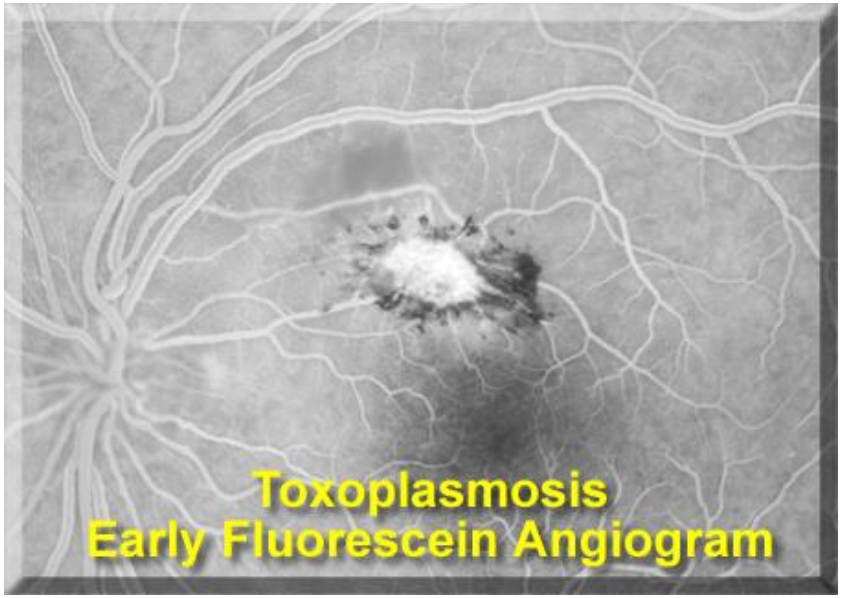
- Most infections produce no symptoms. The time between e xposure to the parasite and system development is 1-2 week s. The disease can affect the brain, lung, heart, eyes or live r.
- Symptoms in persons with otherwise healthy immune syste ms.
  - Abortion (especially in 1st and 2nd trimester)
  - Fetus can have hydrocephalus
  - Enlarged lymph nodes in the head and neck
  - Headache
  - Mild illness with fever, similar to mononucleosis
  - Muscle pain
  - Sore throat

Symptoms

- Symptoms in immune suppressed persons:
  - Confusion
  - Fever
  - Headache
  - Retinal inflammation that causes blurred vision
  - Seizures

Diagnostic tests and e xpected results

- Antibody titer for toxoplasmosis
- Fecal examination for toxoplasma oocysts
- Cranial CT scan
- MRI of head
- Slit lamp exam
- Brain biopsy
- ELISA
- Fulton test
- Indirect Fluorescent Antibody test



Recommended treatment

- No treatment is required for a healthy person, symptoms go away within several weeks to months.
- Sulfadiazine and Pyrimethamine are used in the acute state of toxoplasmosis.
- In difficult cases spiramycin is used
- Clindamycin is the treatment of choice for dogs/cats.
- Antibiotics do not destroy the infection. It is an infection you live with the rest of your life.

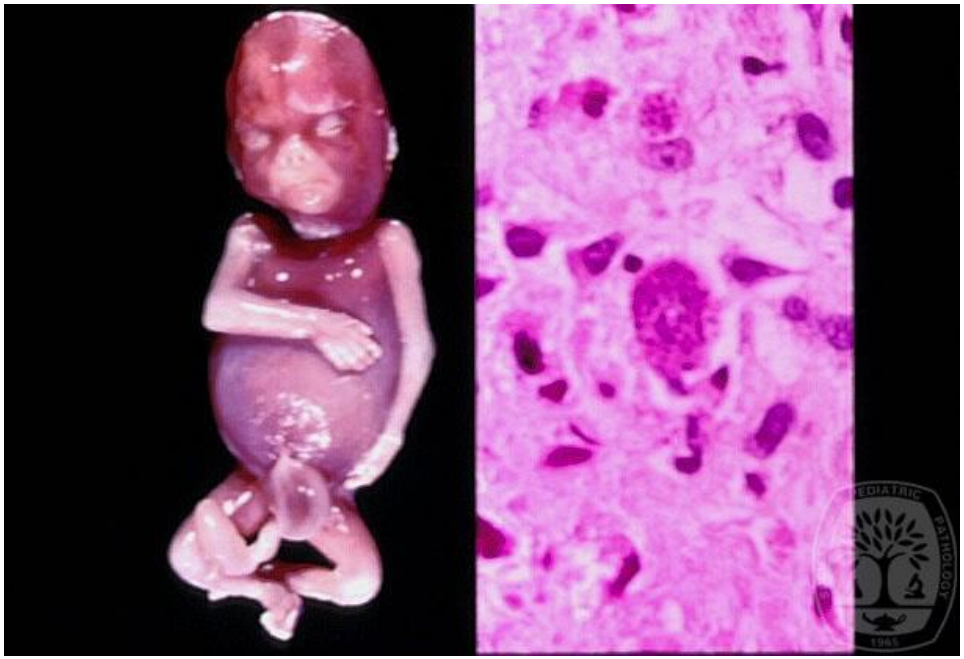
Prognosis

- Acute infection in children may cause swelling of the retina of the eye.
- In cats most have a poor prognosis with a severe i nfection due to the intense dehydration.
- Adults that are healthy have a good outcome.
- Complications may occur with this disease:
  - Personal disabilities such as blindness, learning disord ers in infants with congenital toxoplasmosis.
  - Return of the disease
  - Spread of the infection in a person with a weakened im mune system.

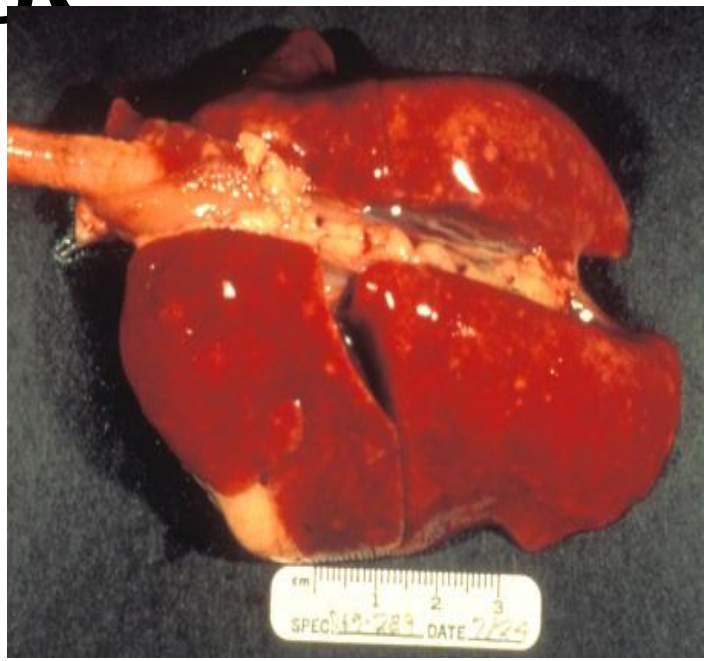
Pathologic lesions of disease

- In tissues rapidly multiplying tachyzoites (trophoz oites) and cysts may be identified. The presence o f tachyzoites is diagnostic of acute infection. Cys ts containing hundreds and **sometimes thousands of bradyzoites make their appearance in brain, skeletal muscles**, and other tissues with the devel opment of immunity.
- Rupture of these cysts has been proposed as a pat hogenetic mechanisms for the development of infl ammatory lesions.
- Lesions can occur in any tissue of the body.

Pathologic lesions of disease

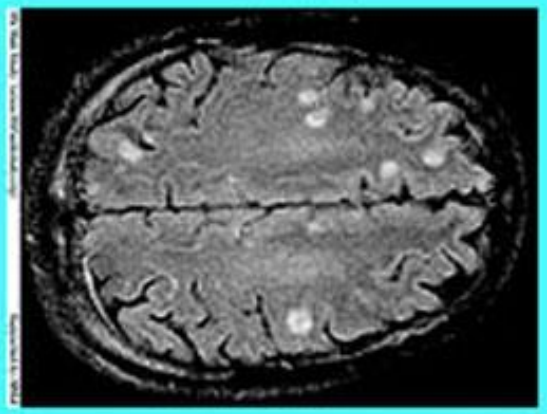


16 week intrauterine fetal demise due to Toxo plasmosis. Organism is preserved despite auto lysis of fetus

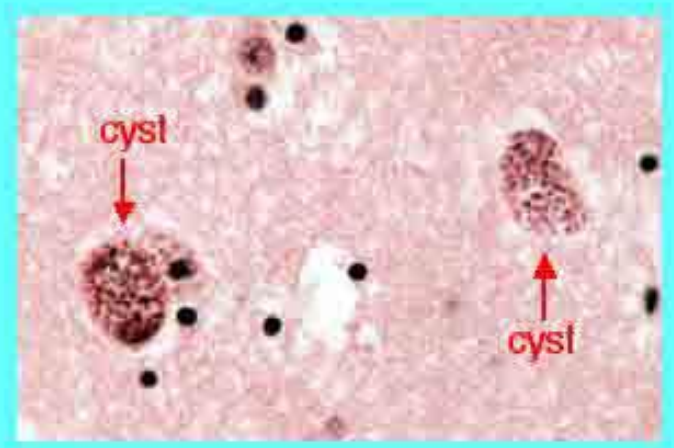


Toxoplasmosis in heart

Toxoplasmosis



Toxoplasma gondii



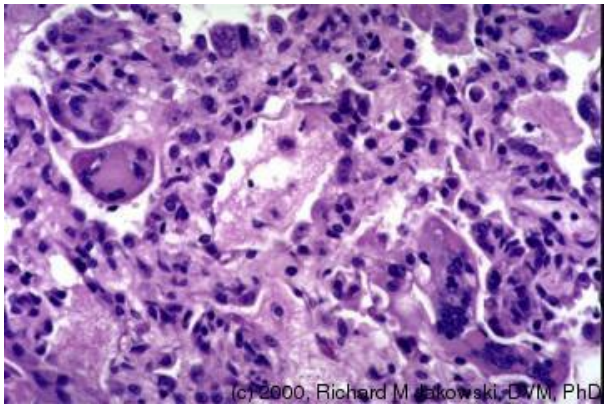
T. gondii bradyzoites
in mouse brain
tissue cysts

T. gondii tachyzoites
in leukocyte

Pathologic Lesions

The first 3 pictures are lesions in the brain and CNS.

The last picture are lesions in a newborn with Toxo



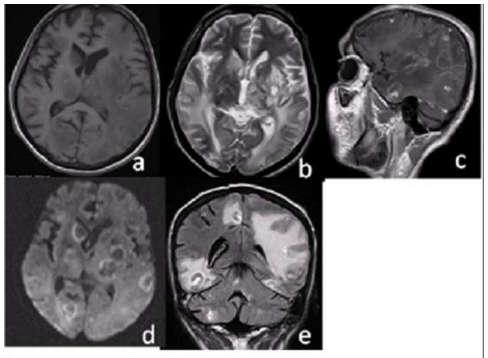
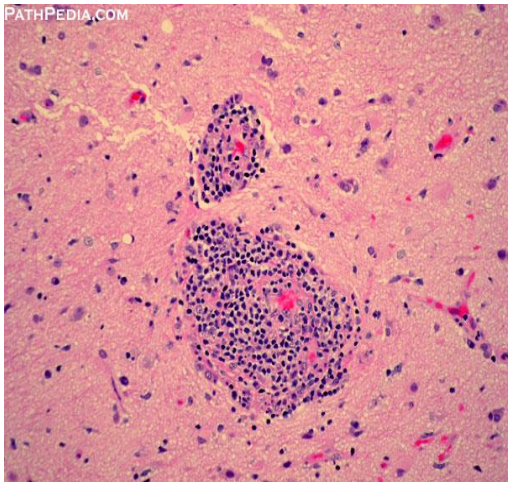


Figure-1: (a) T1-Weighted Axial MR image showing multiple hypointense lesions with perilesional edema. (b) T2-Weighted Axial MR image showing multiple hyperintense lesions of varying sizes with marked surrounding vasogenic edema. (c) Post contrast sagittal T1 image showing multiple lesions with enhancement in the periphery and centre. (d) Post contrast coronal image showing multiple supratentorial and infratentorial ring enhancing lesions with eccentric nodules: the "target sign". (e) Diffusion weighted axial MR image showing multiple ring enhancing lesions of varying sizes.



Prevention

- Wash your hands with soap and water after ex posure to soil, sand, raw meat or unwashed veg etables.
- Cook your meat thoroughly
- Wash and/or peel all fruits and vegetables befo re eating them.
- Wear gloves when gardening or handling sand from a sandbox. Wash hands well afterwards.

Prevention

- Avoid drinking untreated water especially in less devel oped countries.
- If you are pregnant:
  - Have someone else change your litter box if possible. If no t then wear disposable gloves and wash your hands thoroug hly.
  - Change the litter box daily because the parasite does not be come infectious until 1-5 days after it is shed in the feces.
  - Never feed a cat raw meat.
  - Keep indoor cats indoors.
  - Avoid stray cats, especially kittens.

Prevention

- Cover your outdoor sandboxes.
- Do not get a new cat while you are pregnant.

Shigellosis

د/سامية الشطبي

إعداد/

دائرة طب المجتمع – اللجنة العلمية 37A

- في حال وجود أي سؤال أو استفسار يُرجى التواصل معنا عن طريق بوت الدائرة
@Communitymedicine37ABot

- **Types of Diarrheal gut infection:**

1. Secretary diarrheal infection:

- When the bacteria reach to the jejunum → (Lead to) Edematous changes in the jejunum
- This type of diarrhea is painless.
- Example:

Vibrio cholera

Note:
Normal gastric barrier and gastric acid inhibit and kill the bacteria organisms pass to small intestine it will be cause diarrhea disease.

2. Inflammatory diarrheal infection:

- Shigella is one of bacteria that leads to inflammatory diarrhea.
- Colon is the habitat of shigella dysentery.
- Example:
  - *Escherichia coli, Entamoeba histolytica and Campylobacter.*
- Symptoms of inflammatory diarrhea:
  1- Fever.
  2- Bloody diarrhea.
  3- Acute abdominal pain.
  4- Cramping.
  5- Tenesmus.

3. Perforating infection(most dangerous type):

- This infection leads to ileum perforation (Because it's narrow) and invade the blood vessel → (Lead to) bacteremia and toxemia.
- In this case the patient should enter the ICU.

Shigella

- Gram negative bacilli Non spore forming and non-motile.
- There is no carrier in this disease (Why?)
- **Route of infection:**
  - Fecal oral route ⟶ direct.
  - Contaminated equipment towels at schools.
- **Incubation period of disease:**
  - 1 – 2 days.
- **Period of the disease:**
  - 2 weeks.
- **Species of shigella:**
  1. *Shigella dysentery* (most common & more dangerous).
  2. *Shigella flexneri.*
  3. *Shigella sonnei* (less dangerous).
  4. *Shigella boydii.*

- **Shigella dysentery:**

- تكون أكثر خطرا في الأطفال والمسنين وكذلك أصحاب الأمراض المزمنة مثل السكري والأمراض التي تكون المناعة متخصصة وتكون اكثر انتشارًا في أطفال الحضانات والمدارس الداخلية.
- عند حدوث حالات يتم حصر الحالات ومعرفة مكان الإصابة ويتم النزول إلى المطاعم ورياض الأطفال والمدارس ونشوف نظافة دورات المياه وكذلك عمال المطاعم ويتم عمل stool culture لهم.

❖ Shigellosis

- Life threatening in children lead to hemolytic uremic syndrome

♦ Pathogenesis and mode of transmission:

a) Contaminated food.

Eating contaminated food with shigella ⟶ Gastric acid barrier ⟶ Small intestine ⟶ colon —Lead to→ Inflammation of colon —Lead to→ colon ulcer "pathogenic picture of shigellosis"

b) contaminated equipment.

c) Flies, fomites can spread of dysentery infection.

❖ **ملاحظة:**

في حالة قرحة القولون إذا كان المريض immunocompetence يستطيع ان يعمل healing، لكن اذا كان المريض immunocompromised تضل القرحة كما هي.

♦ Epidemiology

- Shigella is caused by bacillary dysentery "the commonest of the unpreventable disease. A world wide problem and an important cause of death and morbidity in young children especially in the third world because of hygiene and the bacterial resistance.
- The number of cases is 200,000,000.
- **Diarrheal disease control program (very important):**
  1) Short term ⟶ خطة أثناء المرض
  2) Long term ⟶ خطة لمنع انتشار المرض
  3) Prevention strategy ⟶ health education, sanitation, disinfection

مثال اذا حصل ٢٠ حاله shigellosis كيف نتصرف ؟

اولاً: يتم حصر الحالات ووضعهم في مستشفى أو مركز صحي ونعالجهم.

ثانيا: منع انتقال العدوى للآخرين وذلك بـ Health education حول المرض.
ثالثا: نعمل لهم الفحوصات اللازمة للتأكد من خلوهم من المرض.

♦ Clinical picture:

a) Fever.
b) Malaise.
c) Acute abdominal pain.
d) Tenesmus.
e) Bloody diarrhea: a. Urgency. b. Sever crumping

اذا كان المريض fulminant خلاص يدخل ICU لأنه في حاله مميتة
Fulminant shigellosis dysentery

♦ Complications

1) Perforation of colon and lead to peritonitis(it is emergency case).
2) Septicemia.
3) Hemolysis uremiC syndrome
4) Toxic encephalopathy.
5) Thrombocytopenia.
6) Intravascular dissemination Coagulopathy.

- هناك نفس الشيء في الenteric fever يؤدي الى perforation التي تؤدي إلى peritonitis
- كل هذه المضاعفات تحصل في immunocompromised

تكون نسبة الوفيات في الامهات الحوامل كثيرة في هذه الحالة لان مناعتهن قليلة وكذلك الاطفال امراض السكري وزارعة الاعضاء وامراض الاورام **(مهم جدا)**

♦ Diagnosis:

1) Clinically.
2) Lab investigation:
   a. CBC
   b. Stool analysis

C. Stool culture.

♦ Treatment

According to patient's state:

1) Fluid replacement
2) Antibiotics:
    a) Ampicillin
    b) Septran (trimethoprim)
    c) Azithromycin
3) Zinc supplement.

❖ If the patient is in emergency room, we should do **ABC**

A* air way

B* breathing

C* circulation

❖ لا نعمل عزل لحالات وباء الشيجيلات إلا إذا كان الوباء endemic.

اللجنة العلمية 37A

CVA

What’s CVA disease ?

It’s group of disease

-coronary heart disease

-Cerebral vascular disease

-Peripheral disease ‘ DVT and pulmonary emboli

-Rheumatic and congenital heart disease ect…

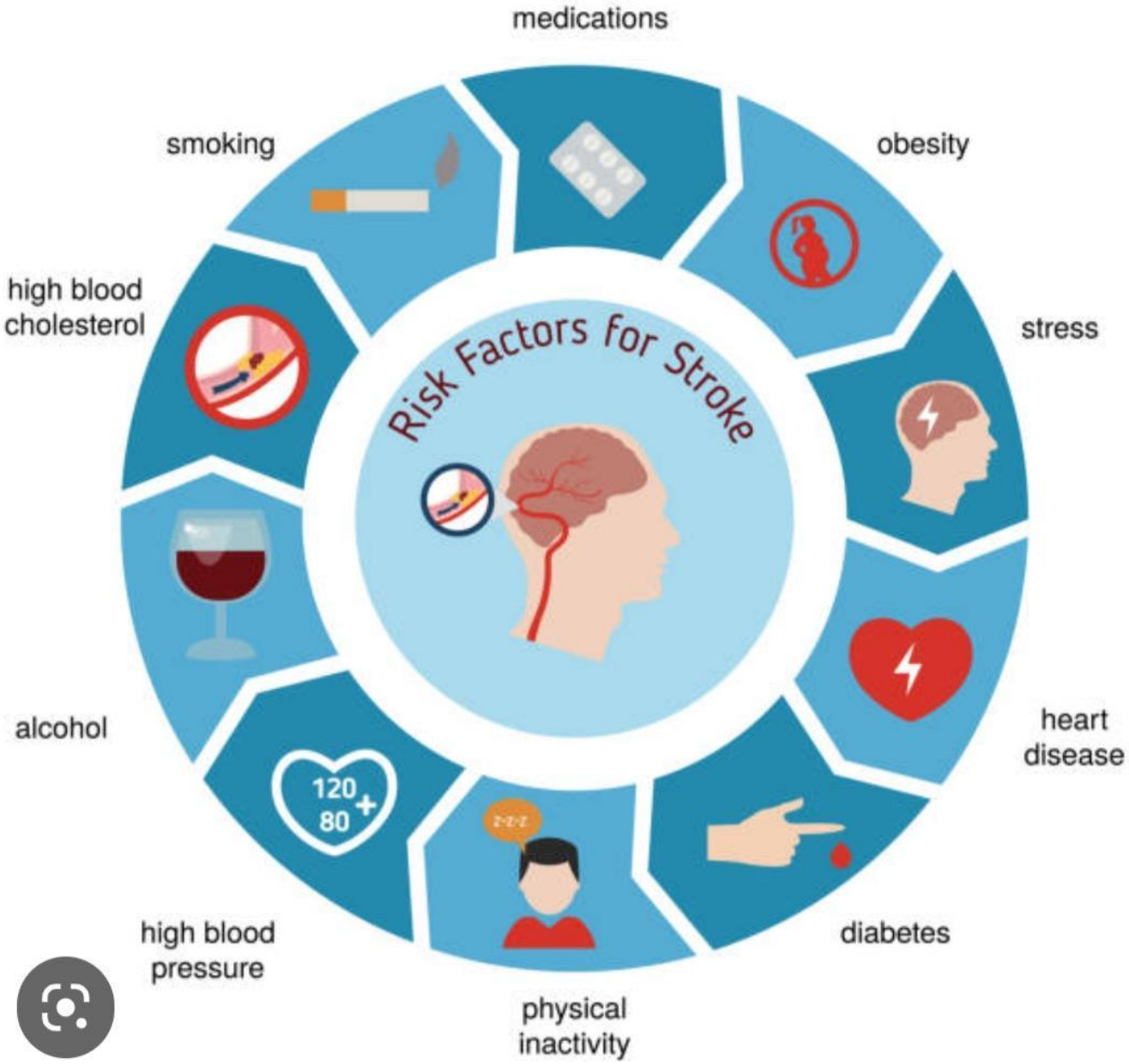
Risk factors of CVA

-modified

-non-modified

-others

-Novel risk factor


medications
obesity
stress
heart disease
diabetes
physical inactivity
high blood pressure
alcohol
high blood cholesterol
smoking
Risk Factors for Stroke
120
80

Framingham point scoring

1. **Age**
2. **HDL**
3. **TC**
4. **Blood pressure**
5. **DM**
6. **Smoker**

Framingham risk score

Decent Score Helping To Save Atherosclerotic Hearts

- DM
- Smoking
- treatment for HTN
- Total cholesterol
- Systolic blood pressure
- Age
- HDL cholesterol

Risk stratification Of cvs:-

1. **Very high risk.**
2. **High risk.**
3. **Intermedium risk.**
4. **Low risk.**

Epidemiology:-

1. **Descriptive study.**
2. **Analytic study.**
3. **Experimental study.**

Prevention of CVA

1-primary

2-secondary.

بسم الله الرحمن الرحيم

EPIDEMIOLOGY OF CARDIOVASCULAR ACCIDENT

Dr. Samaya Al-shatabi

إعداد/

اللجنة العلمية – 37A

في حال وجود أي سؤال أو استفسار

يرجى التواصل معنا عبر البوت

@communitymedicine_37A_bot

❖ What's CVA diseases ?!

- مجموعة من الأمراض التي تصيب الأوعية الدموية ممكن تؤثر على القلب والدماغ...

➢ Coronary heart disease :

- ✓ Myocardial infarction .
- ✓ Ischemic heart disease .
- ✓ Angina .

➢ Cerebral vascular disease

- ✓ Ischemic stroke .
- ✓ Ischemic fraction .
- ✓ Ischemic hemorrhage .

➢ peripheral disease

- ✓ Gangrene in any part in the body → by defect in peripheral blood vessels.
- ✓ DVT & Pulmonary embolism .

➢ Congenital heart disease

- ✓ Atrial septal defect .
- ✓ Ventricular defect .
- ✓ Hyper cholesterol "hereditary ".
- ✓ Hole in the heart .

➢ Rheumatic heart disease → أكثر وفيات الأمهات

- ✓ In some people repeated streptococcus infections cause the immune system to react against the tissues of the body including inflaming and scarring the heart valve .
- ✓ or may lead to valve destruction → lead to MVR &MVS

- هذا يهمني كثير لو انصابت النساء الحوامل بتحدث مشاكل عند الحمل والولادة .

➢ Mortality rate high in :

- Infected valve of the heart .
- MVS .
- MVR .
- HTN .
- Pulmonary oedema .

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➢ Case

❖ **Pregnant women 32years old .she has RHD what happens to to her fetus ?**

- **If Ejection fraction more than 60% low birth weight** →due to hypoxia →placenta insufficiency →decrease nutrient to his fetus.
- **But if Ejection fraction less than 60%** → we will get her an abortion .

- maybe occur preterm or abortion of placenta due to placenta insufficiency → we will give her oxygen until delivery .

Note

- **Most of pregnant women they have RHD rare with stroke, CHD &angina .**

❖ Risk factor of CVD

1. **Modified .**
2. **Non modified .**
3. **Others .**

❖ **Modified**→ **" ممكن اشتغل في المجتمع او غيره "**

1.High blood pressure

- How to prevent from hypertension ?
- How to treat hypertension?
- How to do education ?
- To prevent the complications such as IHD ,stroke and CHD or any problems .

2. abnormal lipid

3. tobacco user or smoker user passive or active

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4. physical activity
5. diabetic
6. obesity
7. unhealthy diet

- مثلا عند الناس الذي يستخدموا السمن بكثرة في اكلهم ويؤدي الى

Increase LDL→ atherosclerosis of vessels →angina or stroke

- Passive يعني لشخص مابيدخن لكن بيجلس مع شخص مدخن .
- Active الشخص المدخن نفسه .
- وكلهم نفس الخطر

❖ Non modified

1. Age
   - average age of person with CVD
   - In male 51---64
   - In female in reproductive health estrogen → increase HDL& decrease LDL
   - BUT in menopause → decrease estrogen → decrease HDL →increase LDL→ atherosclerosis CHD or cerebrovascular disease .
2. Gender male more than female
3. Incidence is the same for both gender

❖ Others

1. Low &middle socioeconomic more dangerous but in high socioeconomic less dangerous→ من أجل برنامج التوعية عندهم قوي
2. Mental health
   - بعض الناس المصابين بامراض نفسية يمكن يموتوا فجاة او الذي ياخذوا علاج الامراض النفسية معرضين للحوادث .
3. Mental illness
4. Psychotherapeutic
5. Heavy alcohol

---

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➢ Searches :

- في عالم جديد اسمه نوفال Noval اخترع اثنين عوامل خطورة :

1. Diabetes hemsystin in blood
2. Inflammation in blood by c-reactive protein
3. Abnormal blood coagulation → increase blood level of fibrinogen→ Clotting thrombosis→ necrosis of blood vessels

Note

- HDL → prevent from stroke
- LDL → increase level of stroke

- RF in pregnancy is more than other people
- Main age of disease 58-50

➢ Physical activity

- ✓ Half hour Exercise
- ✓ Cessation smoking
- ✓ Stop drinking alcohol

➢ Metabolic effect

- ✓ increase tobacco
- ✓ increase blood pressure
- ✓ increase lipid
- ✓ over weight,
- ✓ obesity
- ✓ sometime poverty

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❖ Framingham point scoring → MCQs

1. **Age** → young-- good prognosis
   → Old ---bad prognosis
2. **high density lipoprotein**
   → increase → good prognosis →.decrease → Bad prognosis
3. **TB**
4. **BP HTN or NO**
5. **DM**
6. **smoker**

- اذا عملنا scoringوكان معه عامل خطورة واحد ممكن يتعالج لكن لوكان لديه اكثر من عامل ممكن يموت المريض او يكون عنده عوز بالحركة او قصور بالكلام اوبالسمع
  - **Torming hammering score health to safely from atherosclerosis, heart BP, cholesterol& HTN .**

➢ RISK FACTOR

1. **very high risk factor**
2. **high risk factor**
3. **Intermediated risk factor**
4. **Low risk factor**

- **EXAMPLE :**
  - **Patient with HTN, smoker& old age → very high risk factor**
    " Management to prevent recurrence of angina &stroke "
  - **Patient smoker only → low risk factor**
  - **Patient with DM and complication such as nephropathy ,proteinuria & glomerulonephritis → low risk factor**
  - **Patient with DM &proteinuria → high risk factor**
  - **Shock of any news lead to stroke → low risk factor**

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➢ High risk

- HTN
- Alcoholic
- Smoker
- Diabetic
- Central obesity ”trunk obesity”
- Standard of score

○ SATNDARD OF SCORING

risk fator	Desirable	Borderline	High
Cholesterol	<20 mg	200-239	>240
LDL	<130	130-139	>160
TG	<150mg	150-199	>200
Obesity	18.5-25	52-92	>130
BP systolic /diastolic	120/80	135/81	140/90

❖ Epidemiology

❖ 1.Descriptive study

- دراسة وصفية عن طريق اخذ شخص وادرس عليه هل هو مدخن أولا اوعنده سكر ,دهون اوهو سمين
- أحيانا المكان له دور مثلا سكان حدة غير الأماكن الثانية اغلبيه اكلهم كله دهون

❖ 2. analytic study

- عن طريق دراسة عوامل الخطورة مثلا كم نسبة المدخنين في كلية الطب
- كم نسبة المدخنين في وزارة الاشغال مثلا الموظفين 3000 موظف منهم 500 مدخن
- ثم اعمل لهم فحص الجلوكوز والدهون واعرف هل هذا ممكن يحصل
- امراض القلب والجلطات
- بعدها اخذ عوامل الخطورة الممشروح سابقا واعمل عليه بحث

❖ 3.experiment study

- نشوف مسار المرض كيف وكيف الوقاية منه

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➢ STATISTICE

- In 2016 mortality number in cardiovascular disease 17.9 million most of them stroke and angina
- 31% global death → 85% heart
- Divide from above to below
    1. Stroke &angina in CVD
    2. Cerebrovascular disease
    3. Cerebrovascular accident

- Male more than female
- So we do education programs to decrease mortality rate

❖ How to prevent the risk factor

1. Cessation smoker → stop smoking
2. Decrease salt in diet → especially in person with blood pressure 140/90
3. Decrease unsaturated fatty acid
4. Consumed fruit and vegetables
5. Regular physical activity exercises 30 min
6. Avoidance of alcohol tobacco
7. Treatment of DM, BP, dislipidemia or hyperlipidemia patients and give them antilipid →decrease LDL
8. Health education of school
9. Sleep for 8 hours
10. Drink 8 cups of water

Note

- Epidemiology go out in compaign against smoking → by help smoker by pacemaker in chest to do frequent in chest → to avoid smoking
- لكن بالنسبة للأشخاص المدمنين على التدخين فانه ننصح بممارسة الرياضة
- من المهم عمل برامج في المدارس –> بنصحهم بعدم اكل الدهون والزيوت المشبعة وبممارسة الرياضة والحفاظ عليها.

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❖ PREVENTION

❖ Primary prevention

- by primordial prevention
  - قبل حدوث الكارثة مثل برنامج رياضة اجباري لكل طلاب المدارس وبرنامج الجامعات او نشتغل على المجتمع نقلل عوامل الخطورة والمتابعة

➢ Control risk factor

1. BP check
2. Sports program
3. Health education
4. Lipid profile
5. Glucose profile

❖ Secondary prevention

- By Management in community :

➢ DRUGS

- Aspirin to prevent blood vessels
- Lipid lowering drug “aterovestotine
- B – blocker &ACEI antihypertensive drugs
- If we give him aspirin antilipid, ACEI, b- blocker and stop smoking – 75% success rate
- but if he does not stop smoking – attack after attack until death

➢ Surgery

- Coronary artery bivalve
- Ballon angioplastography
- Valve replacement “prosthatic valve
- Heart transplantation
- Medical device by pacemaker
- Patchy closing to congenital defect

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- Close the opening of heart by this management →I can treat the CVD patients

❖ Tertiary prevention

- The patient in stroke and he cannot walk in this case we will do rehabilitation

➢ EXAMPLE

- angina →breath exercise by long exercise
- Inhalation "oxygen "
- Another case do physiotherapy

❖ QUESTIONS

➢ When does DM lead to death

1. Uncontrol
2. Hypertension
3. Smoker
4. Obesity or over obesity
5. Proteinuria→ nephropathy

➢ How smoking lead to death ?!

- Nicotinic acid in lung → narrowing in blood vessels → Lead to
  - → Insufficiency of oxygen → "hypoxia "
  - → Deposit of cholesterol in blood vessels →Lung insufficiency

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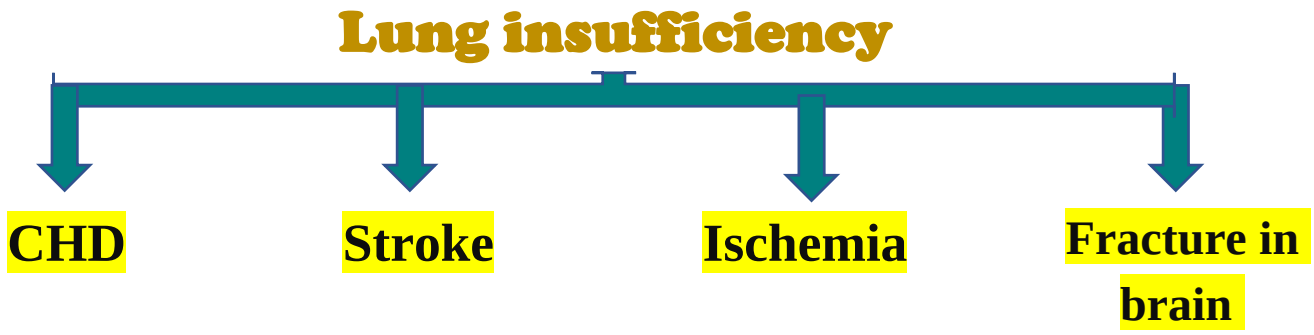

- **If the patient with hypercholesterolemia what is the management**
  - We give him antilipid drugs
  - Name of drugs not important in exam or answer with any drug of family .

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بسم الله الرحمن الرحيم

EPIDEMIOLOGY OF URINARY TRACT INFECTIONS

Dr. Samaya Al-shatabi

إعداد/

اللجنة العلمية – 37A

في حال وجود أي سؤال أو استفسار

يرجى التواصل معنا عبر البوت

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Epidemiology of urinary tract infection

- Definition
- Pathogenesis
- Etiology
- Risk factor
- Classification
- Clinical picture
- Treatment

❖ Definition

- Inflammation of urinary system
- Or It is a Secondary disease Invasion of urinary system by microbe.

❖ note

- Most common infections :
  1) respiratory tract infections
  2) urinary tract infections
- E.coli is most Common among women
- Common in female, children, old age male &pregnant women

urinary tract system is sterile so any foreign body cause infections .

- Why is UTI most common in female more than male?

➔ Shortness of urethra in female

❖ terminology

- bacteriuria: presence of detectable bacteria in urine , Seen in pregnancy
- pyuria: presence of pus cell in urine , Seen in pregnancy
- Bacteria without pyuria: Seen in DM ,enteric fever& bacteria endocarditis

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- Sterile pyuria: presence of plenty pus cell in urine but lack of growth on culture
- urosepsis: defined as threating organ dysfunction Caused by dysregulated host response
- to infection originating from Urinary tract
- It is fatal condition Seen in TB & schistosomiasis

❖ Pathogenesis

➔ Mode of transmission

1) A ascending Route ascending of normal flora or bad hygiene :-
- Urethritis
- Vaginitis
- Cystitis

- **There's pain sensation frequent and dysuria**
- **Frequency permanent micturition (contraction of Urinary bladder)**
  - Mild every half hour .
  - Moderate every 10 minutes .
  - Severe all day .

2) descending route hematogenous
- **Rare due to bacteremia Caused Commonly by :**
  1) staphylococcus aureus
  2) salmonella
  3) Mycobacterium tuberculosis
  4) leptospirosis
  5) candidiasis

❖ Note
- No reflex in urine in adult but there is in children and pregnant women because of compression by uterus
- But If reflex occurs in adults lead to
  1) Dialysis
  2) Pyelonephritis

في هذه الحالة يقومون أحياناً بعمل تصريح جراحي من أجل منع حدوث فشل كلوي

immunity medicine

❖ Etiology

- **Usually bacteria agent**
  - E coli 80% most common cause MCQ
  - Klebsiella
  - pseudomonas aeruginosa
  - enterobacteria
  - Staphylococcal
  - mycobacterium
  - chlamydia
- **fungal such as** :
  - candidiasis albicans
    - ➔ Occurs in :
      - immunocompromised patient .
      - Chemotherapy.
      - Immunosuppressant drugs.
      - In sexual active women lead to recurrent infections
        - ✓ **Treatment of sexual active woman**
          - Antifungal such as fluconazole
          - Cream
          - Douche
          - Good hygiene
- **Parasite**
  - Schistosome haematobium ➔ lead to hemorrhagic cystitis
  - Complaints of patient bloody urine
- **virus**
  - Cytomegalovirus
  - adenovirus
  - HSV

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❖ Risk factor

1) Gender women
2) Old age
3) pregnancy because ➔she is low immunity labilty to fungal infections
4) Menopause ➔ atrophy in vagina wall

5) Infection of urethra ascending cystitis
6) Instrumentation Catheterization such as :
    - Hemiplegia
    - CBI
    - Spinal cord injury
7) Structural obstruction
8) prostatic hypertrophy
9) urethral stricture
10) stone in ureter (functional obstruction)
11) renal transplantation
12) Neurogenic bladder such as CVA problem in spinal cord (no sensation in bladder)
13) Metabolic disorder such as DM
14) Bacteria virulence
15) genetic factor

❖ Classification of UTIS

I. based on Anatomical classification

- Lower UTI urethritis cystitis prostatitis
- Upper UTI acute pyelitis Acute pyelonephritis

II. Basel on source infection

- Community acquired
- Hospital acquired

III. Classification on clinical type.

1) **uncomplication UTI** infection of structure or function normal .
يشتكي المريض من عرض ونعالجه ويتعافى .
2) **Complication UTI** infection in structuring of function abnormal .
يتعالج ويستمر أسبوعين في أخذ المضاد الحيوي .
3) **Recurrent UTI**
    - recurrent of complications or incomplete frequency of least 3UTIS /year or twice UTIs or last six months .

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❖ Clinical picture

➢ Symptoms

- **Urethritis**
  - burning sensation in urination
- **Cystitis**
  1. frequency
  2. Dysuria
  3. low grade fever
- **Acute pyelonephritis and pyelitis**
  1. Sever long pain
  2. High grade fever
  3. Sweating
  4. Dysuria
  5. Bloody urine
- **Sexual active women** ➔ Sexual urethral syndrome
  1. Pain
  2. Fever
  3. Irritation

➢ Asymptomatic bacteria MCQ

- Bacteria without symptoms .
- isolation of specific quantitative Count of bacteria ≥ 10^5(Colony forming unit (CFU) irrespective.

نخاف من مخاطرها في النساء الحوامل .

❖ Complications of pregnancy

1) Abortion
2) Premature rupture membrane
3) Preterm lobar
4) Low birth weight
5) Abruptio placentae
6) IGR
7) Toxic preeclampsia
8) IUFD

لذلك من الضروري في كل زيارة أن نعمل لها فحص بول .

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❖ Investigation

1) Urine analysis
2) Urine culture ➔ (CFU) is significant bacteria>105
3) Ultrasound to kidney ureter
4) IV venography
5) CT scan in recurrent infections
6) Cystoscope in recurrent infections or in bloody urine
7) Biopsy
8) PCR

➢ specimen

- Mostly clean -Catch Mid Stream Urine
  - ➔ **In Catheterized patient**
    - By suprapubic aspiration
  - ➔ **In Renal tuberculosis**
    - Early morning urine analysis seen caseous material .
  - ➔ **initial flow in :**
    - urethritis
    - proctitis
    - Acute urethral syndrome

❖ Transport immediate

- ➢ 4h _4c
- ➢ add baric acid more than 4hours

- In all causes or before any surgery Urine analysis and culture very important .

❖ Treatment

- ➢ **Stay hydration**
- ➢ **Alkalization of urine**
  - Drink strawberry and cherry juice to alkalization urine

community medicine

- **Antibiotics**
  - ✓ **In mild case 3days**
    1) Cefixime
    2) Cebox
    3) Hyaluronic acid
    4) Cephalosporins such as:
       a. Cefotaxime
       b. Ceftriaxone
       c. Microsulfon
  - ✓ **In severe case 1weeks or 2weeks**
    - Determining the cause
  - ✓ **In pregnant women Admission**
    1) Stay hydration
    2) The best analgesic paramol .

❖ **NOTE:**

- In Acute pyelonephritis and pyelitis the best analgesic **aspirin**
- Cystitis the best analgesic **buscopan**

community medicine

Childhood Immunization

DR. NORA AL-AWADHY
PEDIATRICIAN
COMMUNITY MEDICINE DEPARTMENT

Terminology

- Immunization → (It's a host defense against infection)
  - **Active immunity: acquired immunity**

    - Vaccination: antigens administered.
    - Following clinical infection.

  - **Passive immunity: the body doesn't produce its own antibodies**

    - Antibodies administered.
    - Short lived immunity given prior to or soon after exposure.

Terminology

Passive immunity differs from Active immunity in the following

Immunity is rapidly established

Temporary immunity (days to months) until antibodies is eliminated from the body.

Terminology

Herd immunity: it's the level of resistance of a community or group of people to a particular disease

large number immunized to break transmission → unimmunized are also protected.

Polio Eradication.

Terminology

- **Vaccine:-** is an immune biological substance designed to produced specific protection against a given disease.
  - **Protective efficacy:** ability to protect against disease.
  - **Vaccine failure**: disease occurs despite vaccination. High with BCG & pertussis.

Cold Chain

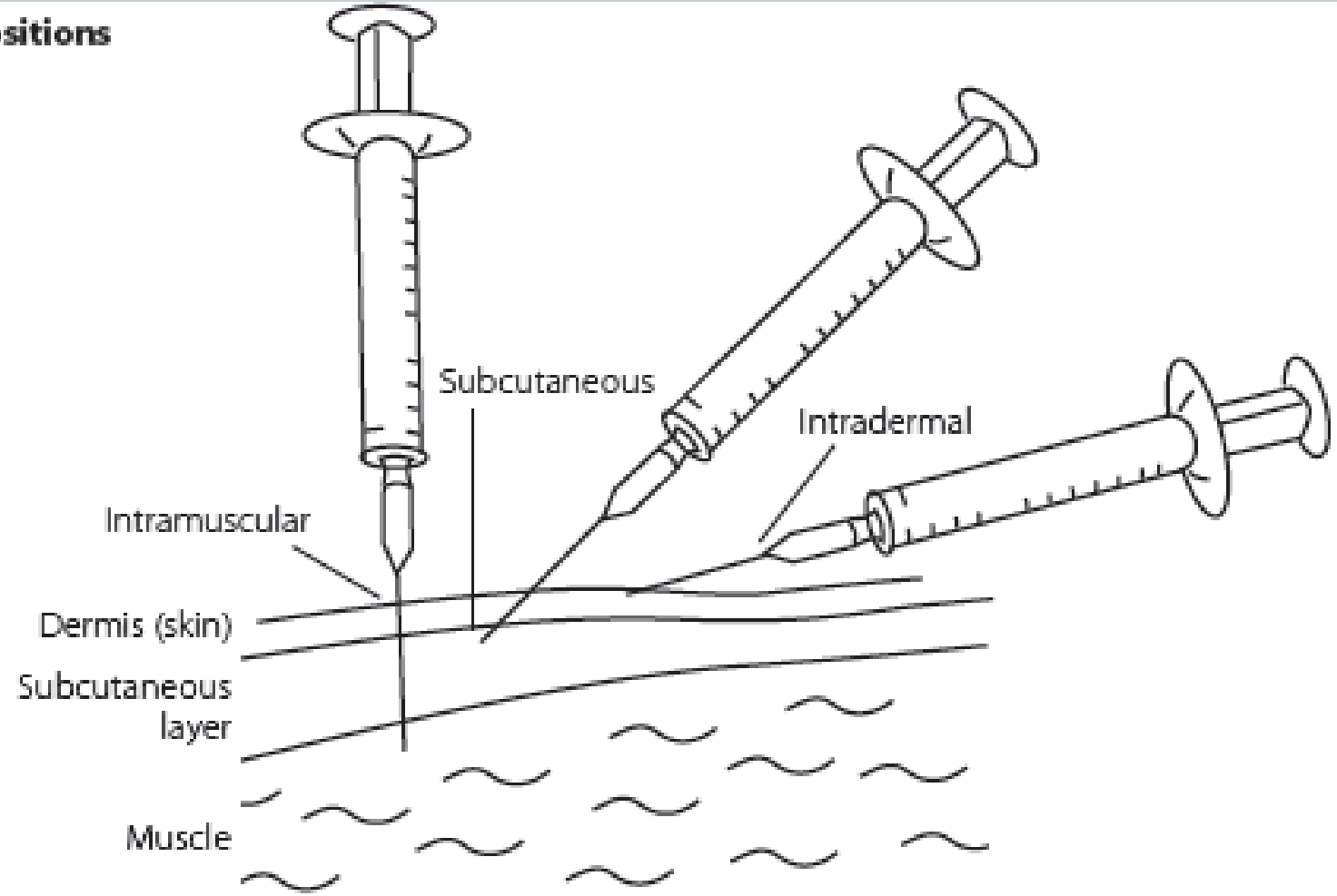
- Proper transport,
- storage, distribution of vaccines.
- At recommended temperatures.

Types of vaccines

- Live vaccines (live attenuated organisms):-
  - **Bacterial: BCG.**
  - **Viral: OPV, measles, rotavirus.**
- Killed (inactivated) vaccines:-
  - **Bacterial: pertussis.**
  - **Viral: inactivated polio vaccine (IPV), rabies.**
- Toxoid (modified toxins): **tetanus, diphtheria.**
- Extracted cellular fraction :
  Bacterial capsular polysaccharide conjugate: Hemoph influenzae type B (Hib), pneumococcal, meningococcal. Viral: hepatitis B (surface antigen).

Ways of vaccine injection

Different needle positions



Contraindication of vaccination

- General

Serious allergic reaction (e.g., anaphylaxis) after a previous vaccine dose or a vaccine component

Contraindication of live vaccine

- Immune compromised patient.
- Malignancy and recent chemotherapy
- Pregnant mother or planned for pregnancy within the next 28 days

الإدارة العامة لصحة الأسرة
البرنامج الوطني للتحصين الصحي الموسع

الجمهورية اليمنية
وزارة الصحة العامة والسكان
قطاع الرعاية الصحية الأولية

بطاقة التحصين
VACCINATION CARD

الاسم:

تاريخ الميلاد: / / محل الميلاد:

المرفق الصحي:

المديرية: المحافظة:

تاريخ الإصدار:

رقم الصفحة الرقم المتسلسل

رقم البطاقة :

إحرص على حصول طفلك على شهادة ميلاد من فرع (مصلحة الأحوال المدنية)

إرشادات هامة

استكمال جميع جرعات التحصين

يحمي الطفل من الأمراض الفتاكة (السل ، شلل الأطفال ، السعال الديكي الخانوق ، الكزاز ، إلتهاب الكبد البائي إلتهاب السحايا ، الإلتهاب الرئوي الحصبة والحصبة الالمانية والإسهال الفيروسي «الروتا»

لا داعي للقلق في حال اصيب الطفل بأعراض الحمى وتورم موضع الحقن فهي اعراض تدل على تفاعل الجسم مع اللقاح ، ويمكن إستخدام الكمادات الباردة على موضع الورم

هذه البطاقة مهمة جداً يجب الحفاظ عليها في مكان آمن ، وعند أي زيارة للطفل للمرفق الصحي ، يجب إحضارها ، وكذا عند دخوله المدرسة

- يرجى الحرص على تحديد عمر الطفل بدقة.

Vaccination schedule

Bacille Calmette-Guérin vaccine (BCG)	**birth;**	
OPV	Oral polio vaccine	birth; 6, 10, 14 weeks;
DTwP,Hib,HepB	Diphtheria and tetanus toxoid with whole cell pertussis, Hib and Hep.B vaccine	6, 10, 14 weeks;
Rotavirus	Rotavirus vaccine	6, 10 weeks;
Injec. polio	In activated polio vaccine(IM inj)	10, 14 weeks;
Measles	Measles vaccine	9, 18 months;
Pneumo_conj	Pneumococcal conjugate vaccine	6, 10, 14 weeks;
TT (women)	Tetanus toxoid	1st contact at pregnancy; +1 month; +6 month; +1 year; +1 year;

جدول تحصين الأطفال

بعد الولادة مباشرة	الزيارة الأولى		لقاح السل جرعة واحدة	
			لقاح الشلل فموي الجرعة التمهيدية	
في عمر شهر ونصف	الزيارة الثانية	الجرعة الأولى	لقاح الشلل فموي	اللقاح الخماسي
			لقاح المكورات الرئوية	لقاح الروتا
في عمر شهرين ونصف	الزيارة الثالثة	الجرعة الثانية	لقاح الشلل فموي	اللقاح الخماسي
			لقاح المكورات الرئوية	لقاح الروتا
في عمر ثلاثة أشهر ونصف	الزيارة الرابعة	الجرعة الثالثة	لقاح الشلل فموي + حقن	
			اللقاح الخماسي	لقاح المكورات الرئوية
في عمر تسعة أشهر	الزيارة الخامسة	الجرعة الأولى	لقاح الحصبة والحصبة الألمانية	لقاح شلل فموي	فيتامين (أ) ١٠٠ الف وحدة دولية
في عمر ١٨ شهراً	الزيارة السادسة	الجرعة الثانية	لقاح الحصبة والحصبة الألمانية	لقاح شلل فموي	فيتامين (أ) ٢٠٠ الف وحدة دولية
من عمر ١٢ شهر إلى ٥٩ شهر	الزيارة السابعة	جرعة تنشيطية من لقاح الخماسي			
من عمر ٥ الى ٧ سنوات	الزيارة الثامنة	جرعة تنشيطية من لقاح الثنائي			

يجب إستكمال جميع جرعات التحصين والحرص على أخذها كاملة في المواعيد المحددة

BCG

- Bacille Calmette-Guérin.
- Live attenuated T.B Bacilli
- Protective efficacy against severe or milliary TB and

TB meningitis= 80%,

against pulm TB= 50% .

- 0.05 ml in neonates(0.1 ml in elders)
- Intra-dermal in right upper arm.

BCG

- Bacille Calmette-Guérin.
- In the 1st 3 months
- At beginning of every school period for tuberculin –ve as **Booster doses**
- **Reaction** Small papule which crust then disappear in 8-12 weeks leaving permanent scar.
- **Complications**
- Regional (axillary)lymphadenitis -need INH
- Abscess formation
- Disseminated infection if given to immunodef. Or improper attenuation

BCG

- **contraindications and precautions**

Contraindications

- Serious allergic reaction (e.g., anaphylaxis) after a previous vaccine dose or a vaccine component

Precautions: Moderate or severe acute illness with or without fever

Primary Vaccination Site Reaction

BCG

- **Predisposing risk factors of TB in Yemen**
- **General risk factors:**

Malnutrition

Poverty

Contact with open cases

Socioeconomic status

Over crowding

Bad ventilation

Chronic diseases as DM

- **Specific risk factors in Yemen:**

OPV

Administration summary: OPV

Type of vaccine	Live oral polio vaccine (OPV)
Number of doses	Four in endemic countries (including birth dose)
Schedule	At birth*, 6, 10, 14 weeks
Booster	Supplementary doses given during polio eradication activities
Contraindications	None
Adverse reactions	VAPP very rarely (approximately 2 to 4 cases per million children vaccinated)
Special precautions	Children known to have rare congenital immune deficiency syndromes should receive IPV rather than OPV.
Dosage	2 drops
Injection site	–
Injection type	–
Storage	Store between 2°C and 8°C (maybe frozen for long-term storage)

OPV cont.

- **Supplementary immunization with OPV:**
  - a key strategy for polio eradication.
  - conducted in large scale campaigns.
  - two doses of OPV, one month apart.
  - all children under five years of age regardless of previous doses.
- If a child has diarrhea, give extra 5^{th} dose of OPV >/= 4 weeks after last dose in schedule.

DTP-HepB+Hib vaccine

Protects against five diseases:,

- Diphtheria
- Tetanus,
- Pertussis,
- Hepatitis B,
- *Haemophilus influenzae type b*.

Administration summary: DTP-HepB+Hib combination vaccine

Type of vaccine	Pentavalent vaccine
Number of doses	Three
Schedule	6, 10, 14 weeks of age
Booster	None
Contraindications	Do not use as a birth dose
Adverse reactions	Mild local and systemic reactions are common
Special precautions	Do not use as a birth dose, usually not given over 6 years of age
Dosage	0.5 ml
Injection site	Outer mid-thigh
Injection type	Intramuscular
Storage	Store between 2°C–8°C. Never freeze

Rotavirus vaccine

- Live oral vaccine (Rotarix).
- Two doses (1.5 ml orally):-
  - 1st at 6wks, no later than 12 wks.
  - 2nd at 10 wks, no later than 24 wks and at least 4 wks from 1st.

Pneumococcal conjugate vaccine

- A capsular polysaccharide conjugate.
- Protects against most *S. pneumonae* infections:
  - Pneumonia
  - Otitis media
  - Meningitis
- 0.5 ml IM left outer mid-thigh: 6, 10, 14 wks.

Measles vaccine

Type of vaccine	Live attenuated viral
Number of doses	2 doses: 9 and 18 month
Contraindications	Severe reaction to previous dose; pregnancy; congenital or acquired immune disorders (not HIV infection)
Adverse reactions	Malaise, fever, rash 5–12 days later; idiopathic thrombocytopenic purpura; rarely, encephalitis, anaphylaxis
Special precautions	None
Dosage	0.5ml
Injection site	Outer mid-thigh/upper arm depending on the age
Injection type	
Storage	Store between 2°C–8°C (vaccine maybe frozen for long-term storage but not the diluent)

Tetanus toxoid immunization schedule for routine immunization of pregnant women

Dose of TT or Td	When to give	Expected duration of protection
1	At first contact or as early as possible in pregnancy	None
2	At least 4 weeks after TT 1	1–3 years
3	At least 6 months after TT 2 or during subsequent pregnancy	At least 5 years
4	At least 1 year after TT 3 or during subsequent pregnancy	At least 10 years
5	At least 1 year after TT 4 or during subsequent pregnancy	For all childbearing years and possibly longer

Contraindications to immunization

- There are no many contraindications to immunization. All infants should be immunized except in these three rare situations:
  1. Anaphylaxis or a severe hypersensitivity reaction is an absolute contraindication to subsequent doses of a vaccine. Persons with a known allergy to a vaccine component should not be vaccinated.
  2. Do not give BCG vaccine to an infant that exhibits the **signs and symptoms** of AIDS.
  3. DPT,Hib,HepB vaccine should NOT be given to children over 5 years of age or to children who have suffered a severe reaction to a previous dose of this vaccine. Instead, diphtheria and tetanus toxoids (DT), and Hep B may be given separately.

Note: an infant with known or suspected HIV infection and/or signs and symptoms of AIDS *should receive measles vaccine at six months and then again at nine months*

Few more notes

Giving vaccines at the same time

If you are giving more than one vaccine, do not use the same syringe and do not use the same arm or leg for more than one injection.

Do not give more than one dose of the same vaccine to a woman or child in one session.

Give doses of the same vaccine at the correct intervals. Wait at least four weeks between doses of OPV, DTP, Hib, and HepB vaccines.

Few more notes

- Delay or lapse → resume from point of interruption, don't repeat schedule.
- Immunization status not known → give age appropriate vaccines.

Rotavirus vaccine

- Live oral vaccine (Rotarix).
- Two doses (1.5 ml orally):-
  - 1st at 6wks, no later than 12 wks.
  - 2nd at 10 wks, no later than 24 wks and at least 4 wks from 1st.
- PE= 92%.

Pneumococcal conjugate vaccine

- A capsular polysaccharide conjugate.
- Protects against most *S. pneumonae* infections:
  - Pneumonia
  - Otitis media
  - Meningitis
- 0.5 ml IM left outer mid-thigh: 6, 10, 14 wks.

Measles vaccine

Type of vaccine	Live attenuated viral
Number of doses	2 doses: 9 and 18 month
Contraindications	Severe reaction to previous dose; pregnancy; congenital or acquired immune disorders (not HIV infection)
Adverse reactions	Malaise, fever, rash 5–12 days later; idiopathic thrombocytopenic purpura; rarely, encephalitis, anaphylaxis
Special precautions	None
Dosage	0.5ml
Injection site	upper arm
Injection type	Subcutaneous
Storage	Store between 2°C–8°C (vaccine maybe frozen for long-term storage but not the diluent)

Tetanus toxoid immunization schedule for routine immunization of pregnant women

Dose of TT or Td	When to give	Expected duration of protection
1	At first contact or as early as possible in pregnancy	None
2	At least 4 weeks after TT 1	1–3 years
3	At least 6 months after TT 2 or during subsequent pregnancy	At least 5 years
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- There are no many contraindications to immunization. All infants should be immunized except in these three rare situations:

١. Anaphylaxis or a severe hypersensitivity reaction is an absolute contraindication to subsequent doses of a vaccine. Persons with a known allergy to a vaccine component should not be vaccinated.

٢. Do not give BCG or yellow fever vaccine to an infant that exhibits the **signs and symptoms** of AIDS.

٣. DPT,Hib,HepB vaccine should NOT be given to children over 5 years of age or to children who have suffered a severe reaction to a previous dose of this vaccine. Instead, diphtheria and tetanus toxoids (DT), and Hep B may be given seperately.

Note: an infant with known or suspected HIV infection and/or signs and symptoms of AIDS *should receive measles vaccine at six months and then again at nine months*

The following are *not* contraindications. Infants with these conditions *should* be immunized:

- allergy or asthma (except if there is a known allergy to a specific component of the vaccine mentioned above);
- any minor illness, such as respiratory tract infections or diarrhoea with temperature below 38.5°C;
- family history of adverse events following immunization;
- family history of convulsions, seizures, or fits;
- treatment with antibiotics;
- known or suspected HIV infection with no signs and symptoms of AIDS;
- signs and symptoms of AIDS, except as noted above;
- child being breastfed;
- chronic illnesses such as chronic diseases of the heart, lung, kidney, or liver
- stable neurological conditions, such as cerebral palsy or Down's Syndrome;
- premature or low-birthweight (vaccination should not be postponed);
- recent or imminent surgery;
- malnutrition; and
- history of jaundice at birth.

Giving vaccines at the same time

If you are giving more than one vaccine, do not use the same syringe and do not use the same arm or leg for more than one injection.

Do not give more than one dose of the same vaccine to a woman or child in one session.

Give doses of the same vaccine at the correct intervals. Wait at least four weeks between doses of OPV, DTP, Hib, and HepB vaccines.

Few more notes

- Delay or lapse → resume from point of interruption, don't repeat schedule.
- Immunization status not known → give age appropriate vaccines.

Post exposure vaccine

- Rabies
- Tetanus

THANKS

Epidemiology and prevention of diarrheal disease + poliomyelitis

Dr. Nora Al-Awadhy

Acute diarrheal disorders among children

Acute respiratory infections, diarrheal disease, malnutrition , malaria, and measles are the most common causes of mortality and morbidity among children in developing countries.

Acute diarrheal disorders among children

Diarrhea is among the greatest killer of infants and young children in developing countries,

Acute diarrheal disorders among children

WHO &UNICF—the principle objectives in assisting many countries to develop and implement integrated management of childhood illness program are:

✓Reduction of mortality and morbidity among young children< 5 years from the following diseases(pneumonia, diarrheal disease, malnutrition , malaria, and measles).

✓ Health Promotion And Preventive Strategies as(immunization, breast feeding, nutritional counseling, vit A supplementation, treatment of worm infestation, growth monitoring and development).

✓Improving the health system.

Definition

- **WHO defines diarrhea** as: the increase of volume, fluidity, or frequency of motions relative to the usual pattern of the individual
- **Persistent diarrhoea** is defined as acute diarrhea (watery **or dysentery) but** persists more than 14 days
- **Chronic diarrhea**: - Diarrhea of gradual onset lasting more or= 14 days or recurrent due to non infectious cause
- The change in stool consistency is more important than stool frequency . This typically translates to a stool volume of more than 10 grams/kg/day in infants and toddlers, or more than 200 grams/day in older children .
- A single loose or watery stool containing blood , pus or mucous in non breast feed babay

Definition

diarrhea in non breast feed baby.

- A single loose or watery stool containing blood , pus or mucous in stool.

diarrhea in breast feed baby.

- Several soft or semi-liquid stools each day or motion that considered abnormal by mother

Prevalence and mortality

- In developing countries, persistent diarrhoea is a common condition.
- Overall, fewer than 10 percent of episodes of diarrhoea persist for two or more weeks, but reports range from 1.4 to 28.4 percent
- worldwide it occurs in up to 3 to 5 percent of the infant population.
- the risk depends on many factors including the initial enteropathogen, nutritional status, geographical location, and socioeconomic conditions .
-

Pathophysiology

- Secretary diarrhoea:

  occurs when pathogens produce toxins that inhibit absorption and result in unopposed large fluxes of fluid and electrolytes into the small intestine that overwhelm the absorptive capacity of the more distal gut. The classic example is cholera and enterotoxigenic E. coli (ETEC) infections.

Pathophysiology

- Osmotic diarrhoea :
- Intestinal villi damage leads to loss of lactase -- accumulation of non-absorbable solutes in intestinal lumen –osmotic load --shift of water to the intestinal lumen-- diarrhea
- This may occur in the small or large intestine. Diarrhoea typically is exacerbated by eating, and stools may be acidic and contain reducing substances. Unabsorbed dietary fat does not exert the same osmotic effect and steatorrhoea is not necessarily associated with diarrhoea.

Pathophysiology

	Secretory diarrhea	Osmotic diarrhea
Volume Effect of fasting Food type	Large Diarrhea continues Unrelated	Small Diarrhea stop Usually related.

Pathophysiology

- In developing countries, most cases of persistent diarrhoea are triggered by infection.
- In some cases, the original infectious agent may be particularly virulent and difficult to clear such as shigella spp.
- Alternatively, the infection may cause intestinal damage that leads to continuing diarrhoea . The most common example of this is lactose intolerance resulting from destruction of the villus tips where the enzyme LACTASE is found.

Pathophysiology

- The third mechanism is delayed healing of the intestine.
- In this scenario, host factors such as zinc or vitamin A deficiency, or generalized malnutrition affect the host response to the infection, resulting in longer duration. Host factors affecting immunity and delaying recovery also occur in immune deficiency conditions, including HIV AIDS.

Pathophysiology

- Exposure to enteropathogens may be so common that new infections with different pathogens occur before the child has had a chance to recover and these sequential acute episodes merge into one episode of persistent diarrhea.

- Infection, malnutrition, and impaired immunity interact and exacerbate one another

Aetiology

- **Acute non infectious diarrhea**

1. Dietitic

o Over feeding

o Under feeding: Starvation diarrhea (scanty, greenish, mucus)

o Bad feeding: - Change in formula type or concentration

- Introduction of new unsuitable food.

o Lienteric diarrhea: Hyperactive gastro-colic reflex motion short after every feed

Aetiology

- **Acute non infectious diarrhea**

2. Drugs

o Prolonged oral antibiotics (e.g. ampicillin)

o Laxatives to the baby or to lactating mother.

3. Parentral diarrhea (2ry gastro enteritis).

o Due to infections outside GIT e.g. otitis media, respiratory infections.

o Possible mechanisms; toxic absorption or reflex gastro intestinal irritation

o The term parenteral diarrhea is no longer used due to possible intestinal infection.

Aetiology

- **Acute infectious diarrhea**
- **(Gastro Enteritis)**
- Gastroenteritis is due to infection acquired through the fecal-oral route or by ingestion of contaminated food or water.
- Mild = 4-6 motions /day
- Moderate = 6-10 motions /day
- Severe > 10 motions /day
- **Viral (60%), Bacterial, Protozoal**
- **Examples**
- - Rota virus.
- - Norwalk like viruses
- - Adenovirus

Aetiology

- **FUNCTIONAL DIARRHEA** :
- Also been termed **chronic nonspecific diarrhea of childhood or toddler's diarrhea**.
- Onset in infancy or the preschool years, and without failure to thrive or a specific definable cause.
- Defined as the painless passage of three or more large, unformed stools during waking hours for four or more weeks.
- Virtually all children develop normal bowel patterns by four years of age

Aetiology

- malabsorption syndromes : – Generalized malabsorption : • Examples : Celiac disease, cystic fibrosis .
- – Selective Malabsorption : •
- CHO malabsorption (lactose) .
- • Bile acid malabsorption. •
- Acrodermatitis enteropathica .
- • Congenital chloride diarrhea .

Aetiology

- **Postenteritis syndrome** — Most enteric infections in otherwise healthy children resolve within 14 days and do not develop into a chronic diarrheal illness.
- However, in a minority of patients, an acute gastroenteritis can trigger persistent diarrhea by causing mucosal damage to the small intestine.
- In children recently treated with antibiotics, Clostridium difficile may cause a colitis characterized by "pseudomembrane" formation.

Aetiology

- **Celiac disease** :
- (also known as gluten-sensitive enteropathy or nontropical sprue) is an immune-mediated inflammation of the small intestine caused by sensitivity to dietary gluten and related proteins in genetically sensitive individuals.
- The disorder is common, occurring in 0.5 to 1 percent of the general population in most countries. Celiac disease often presents as chronic diarrhea, with or without malnutrition, during late infancy or early childhood.

Aetiology

- **Inflammatory bowel disease** — Ulcerative colitis and Crohn's disease are idiopathic chronic inflammatory diseases of the bowel.
- These disorders typically present with gradual onset of chronic diarrhea, with or without blood, from mid-childhood through adulthood.

- **Allergic enteropathy** — An abnormal immune response to food proteins can cause a proctitis/colitis or an enteropathy. The former tends to present as bloody diarrhea and is frequently triggered by cow's milk protein in infants. The latter presents as nonbloody diarrhea and/or failure to thrive.

Aetiology

- **MALDIGESTION OF FAT**
- **Cystic fibrosis** — Cystic fibrosis is the most common cause of pancreatic exocrine insufficiency in children. The disease may present at birth with meconium ileus, or may be suggested later by gastrointestinal symptoms:
- Of fat malabsorption, failure to thrive, rectal prolapse (particularly in the setting of diarrhea) or pulmonary symptoms.

Aetiology

- **Other causes of pancreatic insufficiency** — Other causes of pancreatic exocrine insufficiency include Shwachman-Diamond syndrome (associated with bone marrow failure and skeletal abnormalities), and two rare disorders, Pearson syndrome and Johanson-Blizzard syndrome.
- **CHOLERHEIC DIARRHEA** — Patients who have undergone resection of the terminal ileum have impaired absorption of bile acids. If sufficient bile acids enter the colon, they may cause a secretory diarrhea.
- Similarly, patients who have had a cholecystectomy can develop cholerheic diarrhea because the continuous drainage of bile into the small bowel may overcome the terminal ileum's reabsorptive capacity

Clinical Approach

- The history : •
- Family history .
- • Age at onset .
- • Dietary history and appetite .
- • Temporal relationship : food/ diarrhea.
- • Type of stool and associated symptoms .
- • Growth and development .

Clinical Approach

- – The physical examination :
- • Growth parameters : weight , length , HC . • Nutritional status : signs of malnutrition , wasting .
- • Special signs : – Abdominal distension (malabsorption) . – Dermatitis (allergy , Acroderm. Enteropathica) . – Edema (P C malnutrition , protein - losing enteropathy) .. – Arthritis (inflammatory bowel disease) . – Retinitis , ataxia (Abetalipoproteinemia

Celiac Disease (a 6-year-old child)

Diagnostic Approach

- The first step in the evaluation is to assess the child for dehydration and sepsis. as both require urgent treatment.
- Children should be weighed, measured, and nutritional status assessed.
- All children should be evaluated for non-intestinal infections, including pneumonia, urinary tract infections, sepsis, and otitis media. Treatment of these infections should follow standard protocols.

Diagnostic Approach

- Clinicians should look for comorbid conditions that may be the underlying cause of the malnutrition, such as cerebral palsy, cystic fibrosis, congenital heart disease, HIV disease, and tuberculosis.
- Classify the diahorrea (watery ,bloody).
- Laboratory investigations. When available, it may be helpful to test the stools for the presence of glucose or acidic pH, to culture bloody stools for Shigella and Campylobacter, and to examine stools for parasites including Giardia and Entamoeba histolytica.

Complications of Gastroenteritis

- Dehydration
- Shock
- Acute renal failure (ARF)
- Metabolic Acidosis
- Electrolyte disturbance
- Convulsions
- Bleeding
- Intussusception
- Persistant diarrhea and eventual Malnutration

Management

- Stabilize children with severe intercurrent infection or dehydration, according to standard protocols.
- The main component of treatment is nutritional rehabilitation.
- Breastfeeding is continued when possible, and supplemented with a balanced diet of locally available foods, including an animal protein source such as yogurt, milk, chicken, or egg.
- Many children with chronic enteropathy have a secondary carbohydrate intolerance and respond better to a low-lactose or low-carbohydrate diet.

Manegement

- in children with bloody diarrhea we suggest empiric therapy directed against Shigella
- Antimicrobial treatment for patients with severe symptoms and suspected or proven cholera.
- Antibiotics should be selected based on sensitivities of local strains. If this information is not available,Ciprofloxacin or Ceftriaxone, are recommended.

Management of functional diahorrea

- If the evaluation suggests functional diarrhea, we suggest trials of the following dietary changes:
- Reduce or eliminate fruit juice or other osmotically active carbohydrates. Apple, prune, and pear juice contain sorbitol and have a particularly high osmotic load.
- Liberalize the fat content of the diet to 35 to 50 percent of total calories.

Prevention of gastroenteritis

- Promote exclusive breast feeding for the first 6 months and continued during illness including diarrhea
- Proper weaning
- Rota virus (Rotarix)vaccines
- Hygienic measures

Poliomyelitis

Dr. Nora Al-Awadhy

WHO/5832 AMRO POLIO USA WHD 1995
Dr Jonas Salk who developed the first polio vaccine in 1955.
Le Dr Jonas Salk, "père" du premier vaccin antipoliomyélitique mis au point en 1955.
PLEASE CREDIT PHOTO WHO/USIS

WHO/21848 AMRO POLIO USA WHD 1995
Professional touch - The late Dr Albert Sabin administers his oral polio vaccine.
Un vrai professionnel - le Dr Albert Sabin, disparu depuis, en train d'administrer son vaccin antipoliomyélitique oral.
PLEASE CREDIT PHOTO WHO/PASTEUR MERIEUX

Overview

Poliomyelitis, a communicable disease often called polio or infantile paralysis, is an acute viral disease, **usually affecting children and young adults,** caused by any of three polioviruses, characterized by inflammation of the motor neurons of the brainstem and spinal cord, and resulting in a motor paralysis, followed by muscular atrophy

Overview

* The words are derived from the Greek polio (grey) and myelin (marrow, indicating the spinal cord).
* It is the effect of poliomyelitis virus on the spinal cord that leads to the classic manifestation of paralysis.

Poliomyelitis can be eliminated and eradicated as

- **polio affect human only and no animal reservoir no long term carrier.**
- **there is no cure for polio but it can only be prevented.**
- **The disease causes a lot of disabilities and deaths**
- **Immunity is lifelong**
- **vaccines are available, effective, safe and inexpensive.**
- **The virus cannot survive for a long period outside the human body.**

Because of that, worldwide , the whole strategies of polio eradication established according to that fact .

strategies for polio eradication:

1. **High routine immunization coverage:** High routine immunization coverage will reduce polio to low levels and set the stage for eradication.
2. **National immunization days:** Mass immunization campaigns called national immunization days (NIDs) are used to stop wild poliovirus transmission. WHO recommends that all children <5 years of age be immunized, regardless of Prior immunization status. Two rounds are conducted, normally 4-6 weeks apart.(1996 started in Yemen)
3. **Acute flaccid paralysis (AFP) surveillance:** sensitive surveillance systems capable Of identifying cases of polio be in place in every country. Data from these surveillance systems will also be needed to certify eradication(in Yemen the programme established in 1998 and a national expert committee in 1998)
4. **Mopping up:** Successful NIDs will reduce poliovirus transmission to a few final chains of transmission, typically persisting in densely populated areas where inadequate health services yield both low routine immunization coverage and poor performance during NIDs.

Strategies for Polio Eradication in Yemen :

1.High routine immunization coverage: in Yemen started at 1979 with OPV

2.National immunization days: Mass immunization campaigns called national immunization days (NIDs) . Two rounds are conducted, normally 4-6 weeks apart.(1996 started in Yemen)

3.Acute flaccid paralysis (AFP) surveillance: (in Yemen the programme established in 1998 and a national expert committee in 1998)

4 .Mopping up: national certification committee in Yemen established at 1999..

الجمهورية اليمنية
وزارة الصحة العامة والسكان
قطاع الرعاية الصحية الأولية
الإدارة العامة لمكافحة الامراض والترصد
برنامج ترصد الشلل الرخو الحاد

النشرة الوبائية الاسبوع ٤٨ لبرنامج ترصد للشلل .pdf

WHO Regions

The world is divided to 6 regions:

1. **America** which certified polio eradication in 1991.
2. **European** which certified polio eradication in 1998.
3. **Western pacific** which certified polio eradication in 1997.
4. **Africa .**
5. **South East Asian**
6. **Mediterranean .**

Infectious agents

* Poliovirus is a member of the family Picornaviridae ,enterovirus subgroup.
* There are three well-defined poliovirus serotypes (P1(the most paralytic and epidemic type), P2(uncommon), and P3(less frequent but cause epidemics). There is minimal heterotypic immunity between the three serotypes. That is, immunity to one serotype does not produce significant immunity to the other serotypes.

Reservoir :

Humans are the only known reservoir of poliovirus, which is transmitted most frequently by persons with inapparent infections.

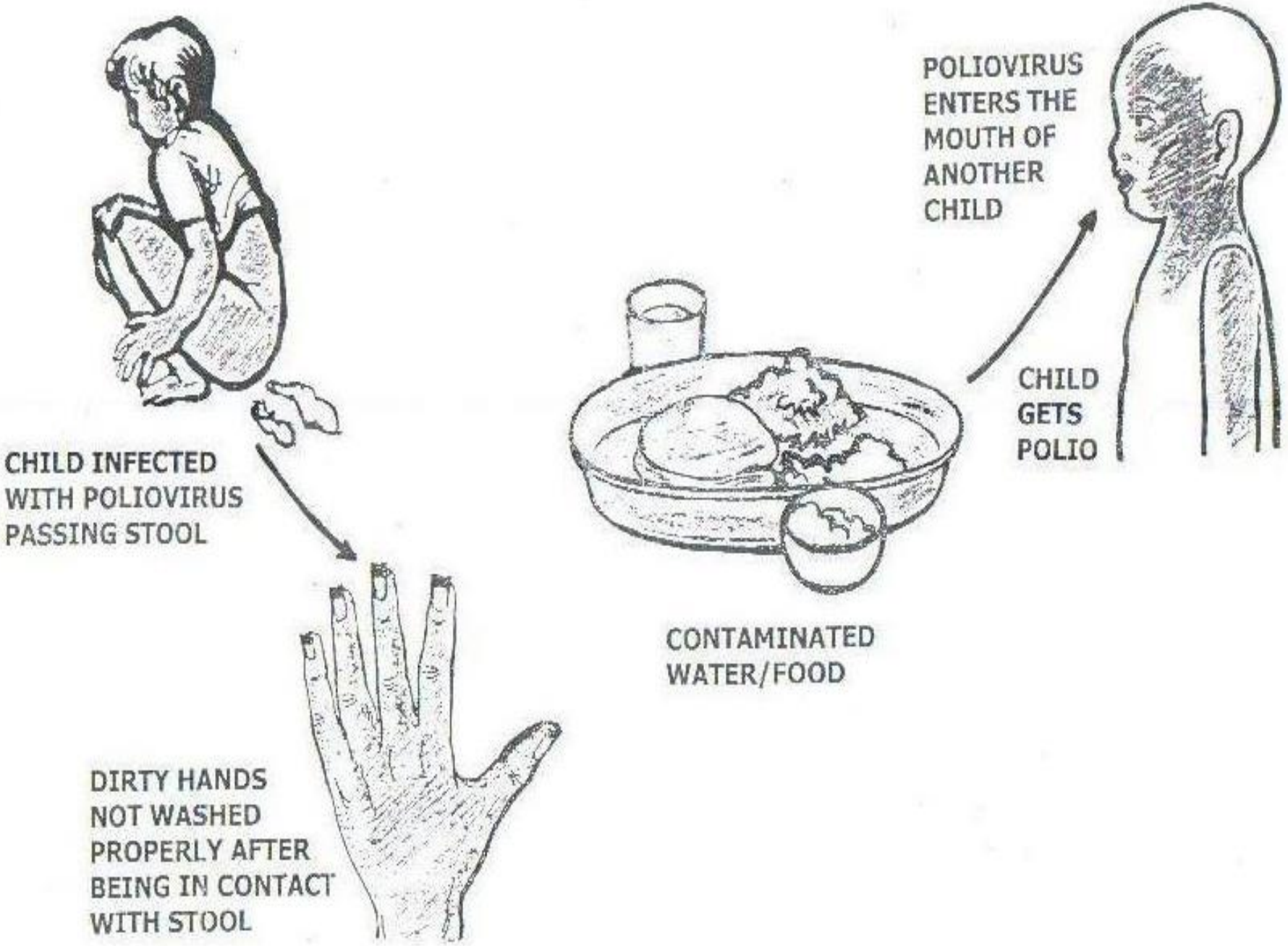
There is no asymptomatic carrier state except in immunodeficient persons.

Who is at risk?

* Polio can strike at any age, but it mainly affects children under five years old. Other several key risk factors have been identified as increasing the likelihood of paralysis in a person infected with polio.

These include:

1. Immune deficiency.
2. Pregnancy.
3. Removal of the tonsils (tonsillectomy).
4. Intramuscular injections, e.g. medications.
5. Strenuous exercise.


CHILD INFECTED WITH POLIOVIRUS PASSING STOOL
DIRTY HANDS NOT WASHED PROPERLY AFTER BEING IN CONTACT WITH STOOL
CONTAMINATED WATER/FOOD
POLIOVIRUS ENTERS THE MOUTH OF ANOTHER CHILD
CHILD GETS POLIO

Transmission :

* Polio is spread through person-to-person contact. When a child is infected with wild poliovirus, the virus enters the body through the mouth and multiplies in the intestine.
* It is then shed into the environment through the faeces where it can spread rapidly through a community, especially in situations of poor hygiene and sanitation

Transmission :

The virus is spread mainly through the **fecal-oral route** or, less frequently, by a **common vehicle** (e.g. contaminated water or food)

Transmission :

* Most people infected with the poliovirus have no signs of illness and are never aware they have been infected. These symptomless people carry the virus in their intestines and can “silently” spread the infection to thousands of others before the first case of polio paralysis emerges.
* For this reason, WHO considers a single confirmed case of polio paralysis to be evidence of an **epidemic** – particularly in countries where very few cases occur.

Communicability :

* Poliovirus is highly infectious, with seroconversion rates among susceptible household contacts of children nearly 100%, and greater than 90% among susceptible household contacts of adults. Persons infected with poliovirus are **most infectious from 7 to 10 days before and after the onset of symptoms**, but poliovirus may be **present in the stool from 3 to 6 weeks.**

Clinical manifestation of poliomyelitis

- The incubation period is 7-14 days. Although polio is essentially a disease of childhood,
- The clinical manifestations vary considerably:
- **Categories of poliomyelitis:**

- ✓ **Asymptomatic poliomyelitis (subclinical)**
- ✓ **Abortive poliomyelitis (nonspecific febrile illness):**
- ✓ **Non paralytic poliomyelitis(aseptic meningitis):**T.
- ✓ **Paralytic poliomyelitis:**

Clinical manifestation of poliomyelitis

1. **Asymptomatic poliomyelitis (subclinical)**:**90-95%** of the infected persons are **asymptomatic.**
2. **Abortive poliomyelitis (nonspecific febrile illness):**Form **4-8% of polio with mild and short coarse.** This type characterized by presence of febrile illness with one or more others symptoms like malaise, anorexia, nausea, vomiting, headache, sore throat, constipation and Un-localized abdominal pain. Usually the fever does not rise above 39.5c
3. **Non paralytic poliomyelitis(aseptic meningitis):**The **poliovirus enter to the nervous system but does not affect or destroy the neurons .**The initial symptoms (called minor illness) are fever ,myalgia, sore throat and headache for 2-6 days .After some days ,signs of aseptic meningitis appear(recurrent fever, headache, neck stiffness, spinal rigidity and nausea).Transient bladder paralysis and constipation may occur .This phase is(called major illness)Tendon reflexes are depressed or exaggerated 8-24 hours after depression of superficial reflexes and indicated impending paralysis . DeepThere is no sensory deficit occurs.
4. **Paralytic poliomyelitis:**This type constitute less than **1% of all type** of polio .In addition to symptoms and signs of non-paralytic polio, symptoms of **generalized weakness of one or more muscle groups** occur.

Paralysis is usually asymmetrical (legs>arms, proximal more than distal) ,rapid onset of flaccid paralysis and become complete when the temperature returned to normal level.
Paralytic polio is classified into types, depending on the level of involvement.

* **Spinal Form :**
* Is the most common type, characterized by weakness of some muscles of neck, abdomen, diaphragm and extremities. Cervical and thoracic spinal cord segments mainly affected.
* There is respiratory insufficiency (tightness, weakness or paralysis of respiratory muscles mainly diaphragm and intercostal muscle.

* **Bulbar Form :**
* Is most common cause of death, characterized by involvement of motor cranial nerve nuclei with or without involvement of vital centers.
* The most nerve affected are 9, 10 and11 cranial nerve and this lead to paralysis of pharynx, larynx and tongue

Bulbar Form :

The clinical finding of bulbar poliomyelitis are:

- Nasal tone to voice or cry.
- Inability to swallowing and accumulation of saliva in pharynx.
- Nasal regurgitation.
- Lack of effective cough.

Deviation of the tongue.

- Hoarseness or aphonia due to paralysis of the cords.

Bulbospinal Form :

There is combined involvement of spinal and bulbar regions.

Encephalitic form:

Its relatively uncommon form, characterized by irritability disorientation, drowsiness and coarse tremors.

Outcomes of poliovirus infection

Outcome	Proportion of cases[1]
No symptoms	90–95%
Minor illness	4–8%
Nonparalytic aseptic meningitis	1–2%
Paralytic poliomyelitis	0.1–0.5%
— Spinal polio	79% of paralytic cases
— Bulbospinal polio	19% of paralytic cases
— Bulbar polio	2% of paralytic cases

Diagnosis:

CDC laboratories conduct testing for poliovirus including:

- **Culture intro type differentiation :**
- PCR
- Serologic testing:
- CSF analysis : Detection of poliovirus in CSF is uncommon.

Differential Diagnosis :

1. **Guillain-Barr syndrome (GBS):**
   presents as an ascending, symmetrical, a reflexic paralysis of distal predominance. It does not causes atrophy or deformities.(motor &sensory)
2. **Traumatic neuritis of the sciatic nerve secondary to IM injections (TNC):**
   presents several days after IM injections with pain and hypothermia in the affected limbs; TM is a flaccid, symmetrical paraparesis with neurogenic bladder and a sensory level.
3. **Transverse myelitis:**Symmetric , non progressive , with sensory level
4. **Other disease misdiagnosed as poliomyelitis were:**osteoarticular trauma, myopathies and dystrophies, viral myositis, acute cerebellitis, retroperitoneal tumors and upper motor neuron syndromes.

MANAGEMENT:

* While there is prevention of the poliovirus, there is no treatment to cure infected children. Treatment is supportive. This mean that the symptoms may be treated to improve infected child comfort and recovery.
* Specific treatment for poliomyelitis determined by infected child's doctor **based on**: Child's age, overall health and medical history, and extent of the disease.

MANAGEMENT:

* **Supportive Treatment:**

- ✓ In abortive form of poliomyelitis, analgesic, sedative, proper diet and bed rest is all that is need. In this case patient can be treated at home.
- ✓ In paralytic poliomyelitis, hospitalization is necessary. Also close observation for respiratory in sufficiency by look carefully for progression of respiratory muscle weakness and assessment of impaired ventilation early.(Physiotherapy after 2-3 weeks & orthopedic consultation).

MANAGEMENT:

* **Supportive Treatment:**
- ✓ Keep airway patent by suction and watch for any respiratory distress. This is especially important in case of bulbar poliomyelitis. To avoid aspiration, head should be low with prone position and face to one side.

MANAGEMENT:

* **Supportive Treatment:**
- ✓ Strict bed rest and a neutral position are very important (can be made with the help of sandbags). In this position of patient, feet should be at right angle, knee slightly flexed, and hip and spine should be straight.
- ✓ Analgesic and mild sedative for relief of pain and restlessness. Analgesic are more effective when given with application of hot packs for 15-30 minutes every 2-4 hours.

Prognosis

The more extensive the paralysis in the first 10 days of illness, the more severe is the ultimate disability.

Everyone infected with the virus does not develop paralysis.

Based on the symptoms their severity, polio infection is categorized into three patterns subclinical, non-paralytic and paralytic infection. **Death occurs due to bulbar type**

THE COMPLICATION

Death occure due to bulbar type.

- **Respiratory failure due to :**
- **Respiratory center paralysis.**
- **Respiratory muscle paralysis.**
- **Hypostatic pneumonia.**
- **Gastrointestinal tract :**
- **Paralysis ileus.**
- **Melena due to single or multiple superficial intestinal erosion.**
- **Acute gastric dilatation.**
- **Urinary (due to urine retention)**
- **Urinary tract infection.**
- **Chronic renal failure.**
- **Mild hypertension (due to lesion of the vasoregulatery center in the medulla and underventilation).**
- **Hypercalcemia, nephrocalcinosis and vascular lesions due to prolonged immobilization.**
- **Death are mainly due to complications as a result of respiratory dysfunction.**

PREVENTION & Control

* Preventive measures as vaccination
* Control of patient, contacts, and environment.(isolation, report , protection of contact, investigation, international measures)

WHO Criteria for polio certification eradication

1. 3 years without any endogenous cases of polio (wild type).
2. Adequate stool sample target 80%.
3. Rate of non polio AFP target 1 or more per 100,000 person aged <15 years.
4. Routine vaccination rate target 80%.

Thanks

الجمهورية اليمنية

وزارة الصحة العامة والسكان

قطاع الرعاية الصحية الأولية

الإدارة العامة لمكافحة الامراض والترصد

برنامج ترصد الشلل الرخو الحاد

النشرة الاسبوعية لترصد الشلل الرخو الحاد للأسبوع 48 -2022م

بلغ اجمالي حالات الشلل الرخو الحاد المكتشفة الى الأسبوع (48) 1234 حالة للعام 2022م.شكل الذكور نسبة 58%

معدل الاكتشاف لحالات الشلل الرخو الحاد8.5 (الحد الأدنى 3 لكل 100,000للاطفال اقل من 15 عام من السكان) للعام 2022م
نسبة كفاية العينات 87%.
سجلت اليمن وباء للفيروس المتحور النوع الثاني cVDPV2 من سبتمبر 2021م وحتى الان وهو الأخطر والاشد شراسة وانتشار وعزل الفيروس من عينات 226 حالة موزعة على 19 محافظات .

حقق برنامج ترصد الشلل الرخو الحاد جميع المؤشرات المعيارية بصورة ممتازة خلال السنوات من 2017-2022م , و لازال البرنامج يعاني من تأخر وصول نتائج العينات المرسلة حيث لم يحقق سوى 25% للعام 2022 مقابل 80% كحد ادنى في المعدل العالمي.

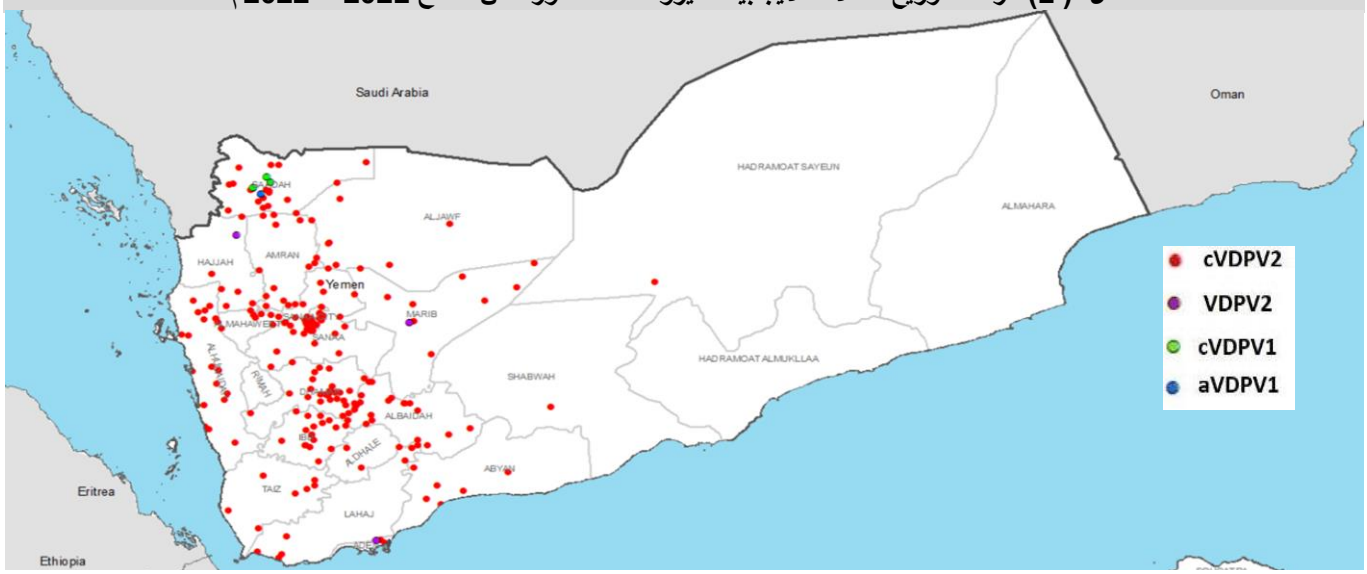
حققت اغلب المحافظات معدل اكتشاف عالي لحالات الشلل الرخو الحاد وبعض المحافظات حققت معدل اكتشاف عالية جدا .
التصنيف النهائي لحالات الشلل الرخو الحاد للعام 2022 م كالتالي158 حالات (13%) فيروسات متحورة من النوع الثاني و 878 (71%) حالة مستبعده و 200 حالة (16%) بانتظار التصنيف .

جدول 1 توزع حالات الفيروس المتحور للنوع الثاني cVDPV2 للعامين 2021- 2022م

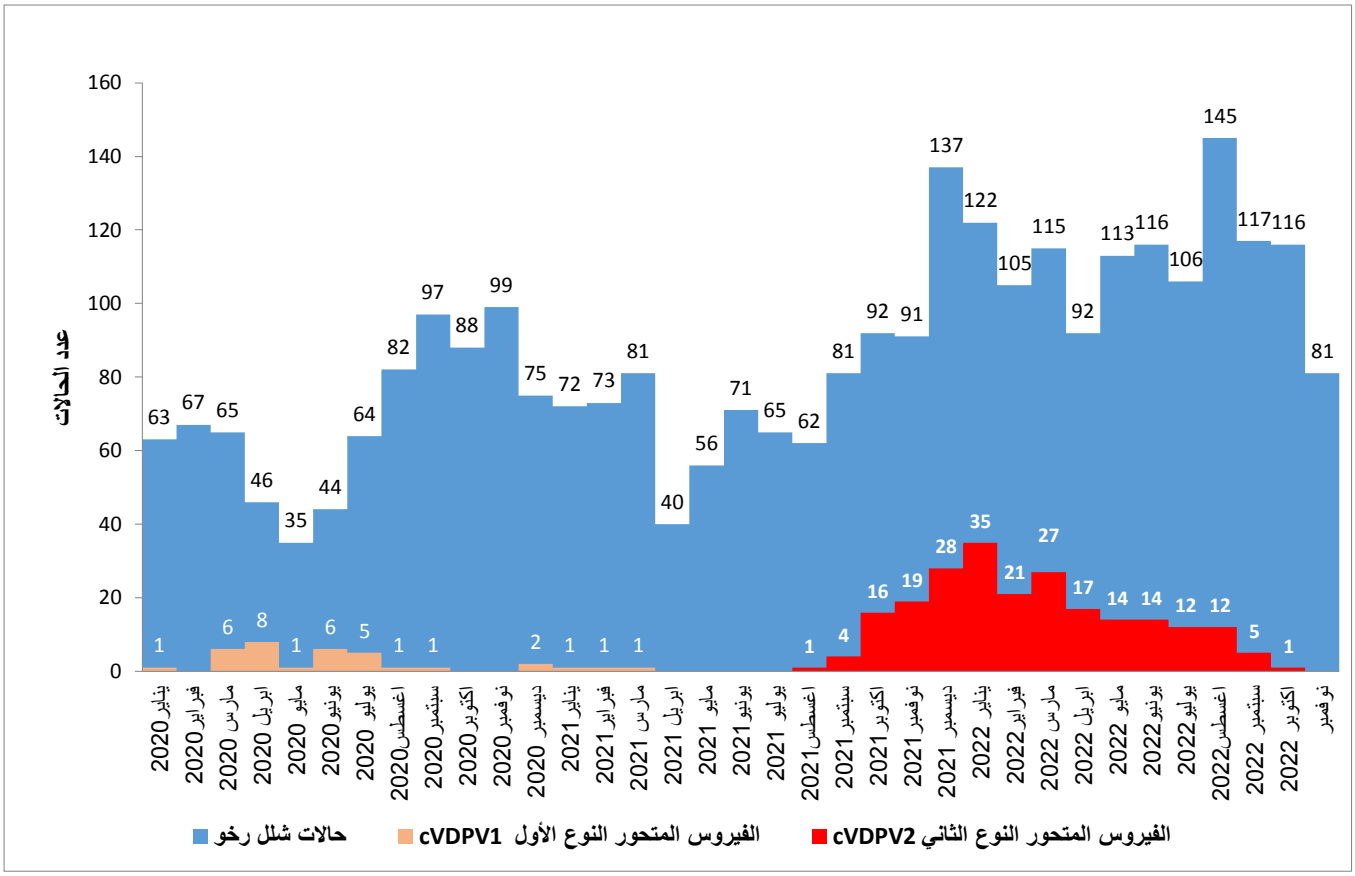
جدول 1 يوضح توزع حالات الفيروس المتحور للنوع الثاني			
المحافظة	2021	2022	الاجمالي
أبين	5	1	6
عدن	3	0	3
البيضاء	1	15	16
الضالع	2	0	2
الحديدة	11	8	19
الجوف	8	4	12
المحويت	0	8	8
عمران	4	9	13
ذمار	2	29	31
سيئون	1	0	1
حجة	1	11	12
اب	0	19	19
لحج	6	3	9
مارب	8	1	9
صعدة	10	7	17
صنعاء	0	24	24
الامانة	0	16	16
شبوة	0	1	1
تعز	6	2	8
الاجمالي	68	158	226

وصلت نتنج العينات البيئية حيث تم تسجيل 13 عينات ايجابية من الفيروسات المتحورة للنوع الثانيcVDPV2 في الاشهر اغسطس 2021- يناير 2022 في الثلاثة المواقع (امانة العاصمة ,عدن ,و الحديدة) حيث ما تزال تتاخر نتائج العينات البيئية من قبل منظمة الصحة العالمية ما تزال 20 عينة بيئية مرسلة لم تصل نتائجها حتى الان

شكل (1)خارطة توزيع الحالات الايجابية للفيروسات المتحورة عن اللقاح 2021 – 2022م



شكل (2) حالات الشلل الرخو الحاد مع الحالات الايجابية من الفيروسات المتحورة من النوع الاول والثاني في الجمهورية اليمنية للاعوام 2020 -الاسبوع 48- 2022م



الشكل (2) المنحنى الوبائي يظهر الارتفاع كبير في حالات الشلل الرخو الحاد في الاشهر يناير - نوفمبر 2022 ولازالت الحالات في تزايد وأيضا ارتفاع عدد الحالات الإيجابية طبقا لنتائج الفحص الواصلة للبرنامج.

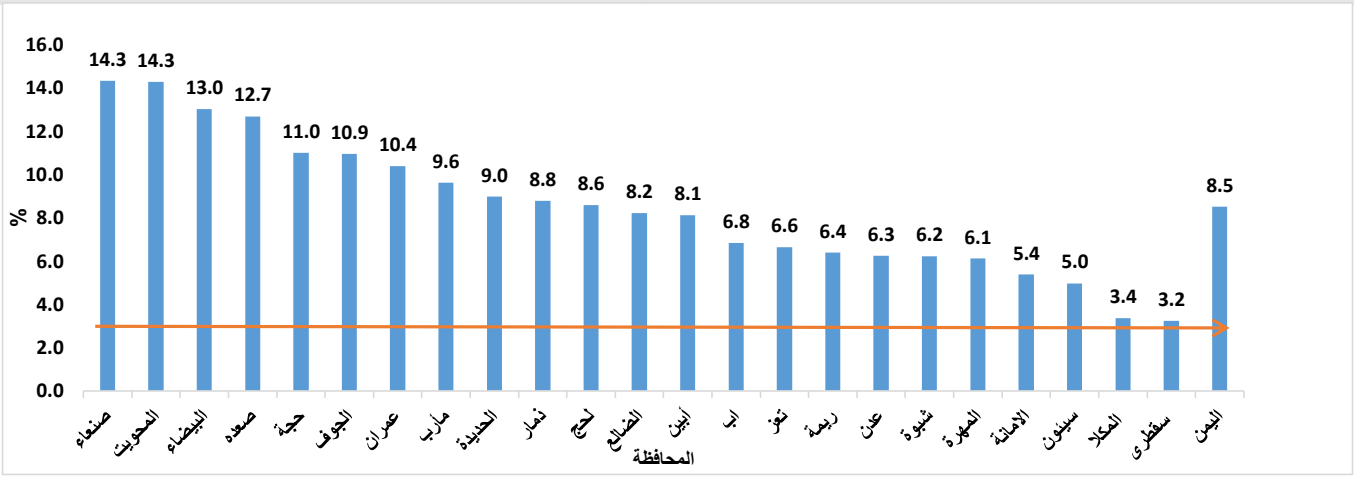
جدول (2) مؤشرات أداء برنامج الشلل الرخو الحاد 2017- الاسبوع48 - 2022

المؤشر\	الهدف	2017	2018	2019	2020	2021	2022
معدل الاكتشاف لحالات الشلل الرخو الحاد 2 /100000<15سنة سنويا	3	5.2	5.1	5.3	5.4	5.9	8.5
نسبة العينات الكافية لحالات الشلل الرخو الحاد	80%	84%	92%	89%	88%	89%	87%
نسبة التقصي خلال يومين من التبليغ	80%	100%	100%	99%	99%	99%	99%
معدل الاكتشاف المبكر للحالات (خلال 7 ايام من بداية حدوث الشلل)	80%	84%	82%	82%	78%	82%	79%
معدل عزل الفيروسات المعوية	10%	16%	29%	18%	18%	21%	*21%
معدل وصول النتائج المخبرية خلال 21يوم من استقبالها	80%	5%	0%	27%	35%	11%	*25%

يلاحظ ارتفاع معدل الاكتشاف لحالات الشلل الرخو الحاد وهذا يدل على تفشات بالفيروس المتحور * حتى الاسبوع 48 حيث تم فحص84% من عينات 2022م

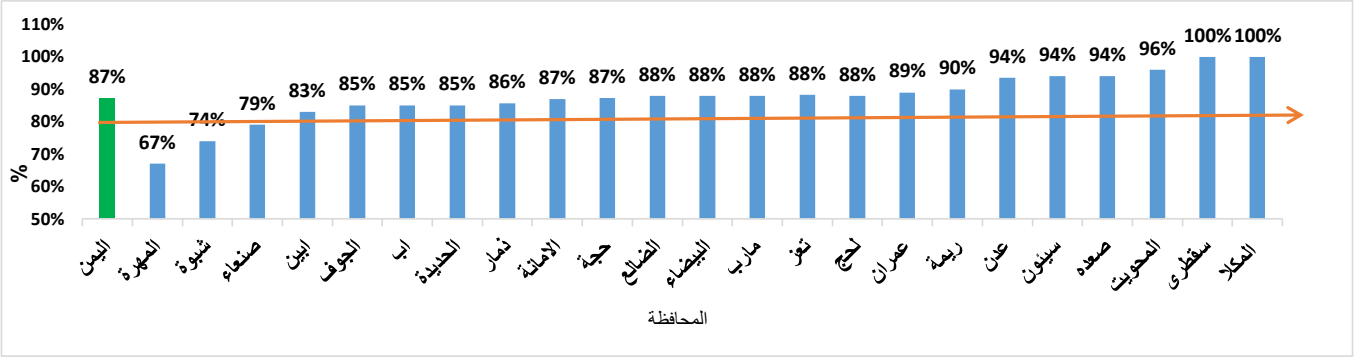
بلغ معدل كفاية العينات 87% في مقابل الحد الادنى وهو 80%
وصل مؤشر التقصي للحالات خلال يومين الى 99%
تدنى مؤشر الاكتشاف خلال 7 ايام لحالات الشلل الرخو الحاد الى 79% وهو اقل من المعدل المطلوب الذي هو 80%
وصل مؤشر عزل الفيوسات المعوية الى 21% مقابل الحد الادنى من ذلك الى 10%
ما زال مؤشر تدني وصول النتائج المخبرية خلال 21 يوما حيث وصل الى 25% من معدل مطلوب كحد ادنى 80%*

شكل (3) معدل اكتشاف حالات الشلل الرخو الحاد للعام 2022 حتى الأسبوع(48) طبقا للمحافظات


16.0
14.0
12.0
10.0
8.0
6.0
4.0
2.0
0.0
%
14.3 14.3 13.0 12.7 11.0 10.9 10.4 9.6 9.0 8.8 8.6 8.2 8.1 6.8 6.6 6.4 6.3 6.2 6.1 5.4 5.0 3.4 3.2 8.5
صنعاء المحويت البيضاء صعده حجة الجوف عمران مارب الحديدة ذمار لحج الضالع أبين اب تعز ريمة عدن شبوة المهرة الامانة سيئون المكلا سقطرى اليمن
المحافظة

يوضح الشكل(3) ارتفاع حالات الشلل الرخو الحاد الكبير في محافظات صنعاء ,المحويت , البيضاء , صعدة , الجوف , حجة ,عمران, مارب ,الحديدة , ذمار , لحج و ابين

شكل (4) معدل ;كفاية عينات حالات الشلل الرخو الحاد للعام 2022 حتى الأسبوع(48) طبقا للمحافظات


110%
100%
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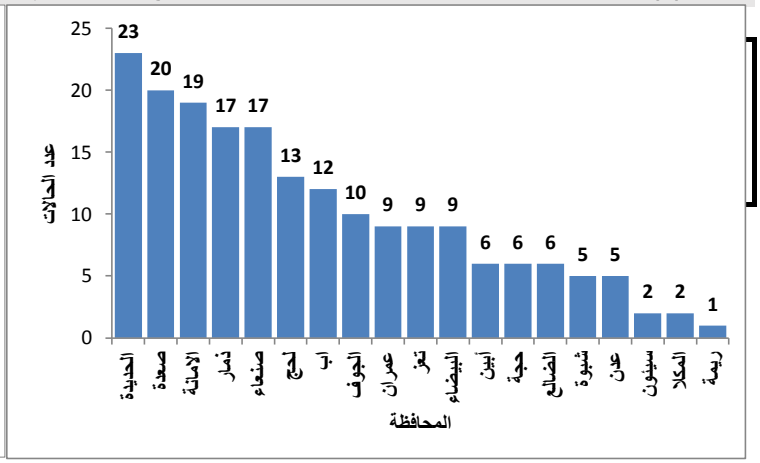
%
87% 67% 74% 79% 83% 85% 85% 85% 86% 87% 87% 88% 88% 88% 88% 88% 89% 90% 94% 94% 94% 96% 100% 100%
اليمن المهرة شبوة صنعاء ابين الجوف اب الحديدة ذمار الامانة حجة الضالع البيضاء مارب تعز لحج عمران ريمة عدن سيئون صعده المحويت سقطرى المكلا
المحافظة

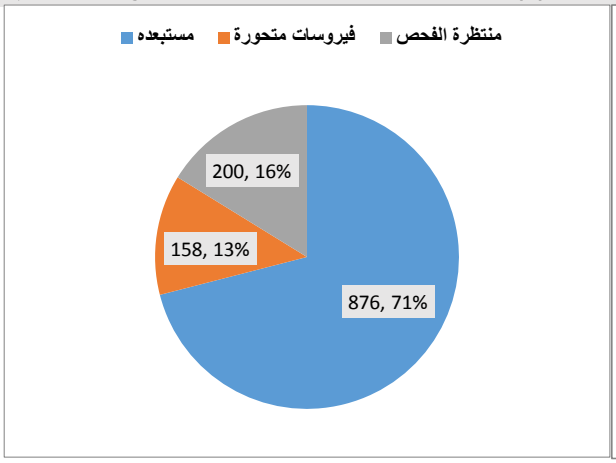
يوضح الشكل 4 تدني كفاية العينات في محافظات شبوة , المهرة وصنعاء

شكل (5) الحالات الصفرية من حالات الشلل الرخو الحاد حتى الاسبوع 48- 2022م


25
20
15
10
5
0
عدد الحالات
23 20 19 17 17 13 12 10 9 9 9 6 6 6 5 5 2 2 1
الحديدة صعدة الامانة ذمار صنعاء لحج اب الجوف عمران تعز البيضاء ابين حجة الضالع شبوة عدن سيئون المكلا ريمة
المحافظة

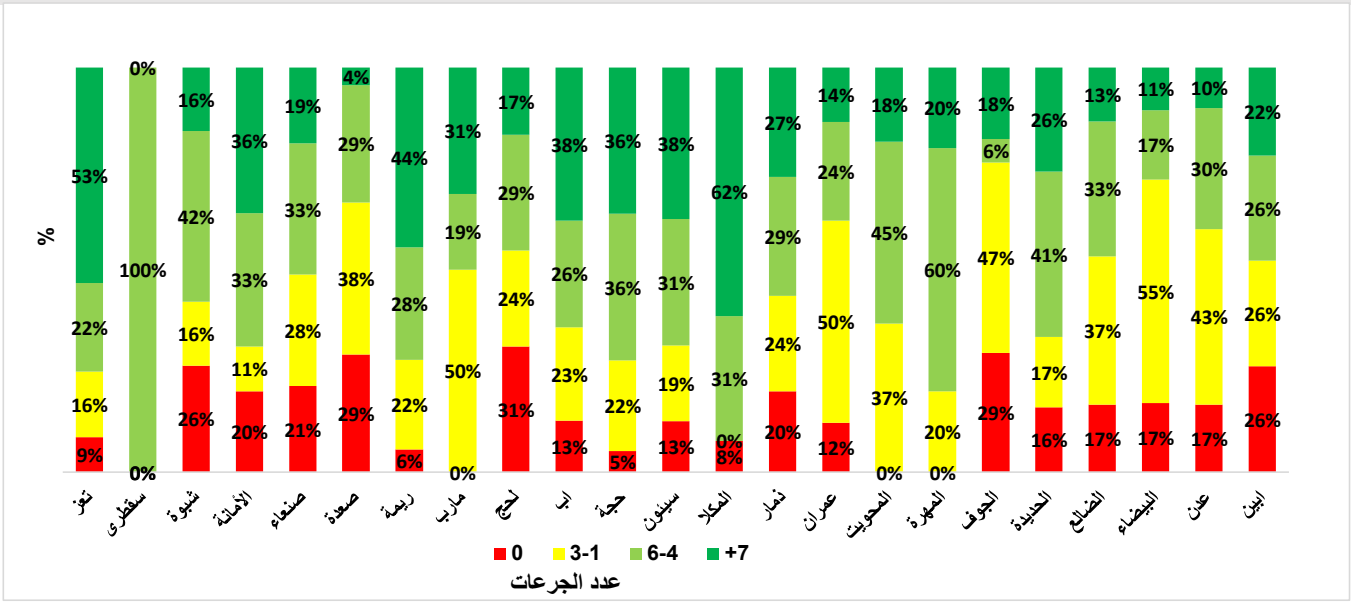
يوضح الشكل 5 عدد الحالات الصفرية (غير مطعمة) عدد191 حالة شلل رخو حاد من 19 محافظة بنسبة (15.4%) كان اغلبها من محافظات الحديدة , امانة العاصمة ,صعدة ,ذمار, صنعاء ,لحج , اب والجوف

شكل (6) تصنيف حالات الشلل الرخو الحاد حتى الاسبوع48- 2022م



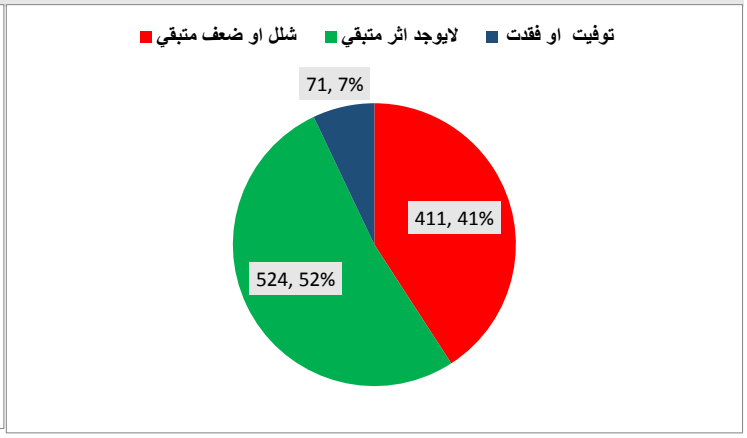
يظهر الشكل (6) لازالت 16% (200) من حالات عام 2022 بانتظار نتائج الفحص , و 71 %(876) سلبية مستبعدة والايجابية للفيروس المتحور 13%عدد (158) حالة

شكل (7) الوضع التطعيم لحالات الشلل الرخو الحاد المكتشفة حتى الاسبوع 48 -2022م



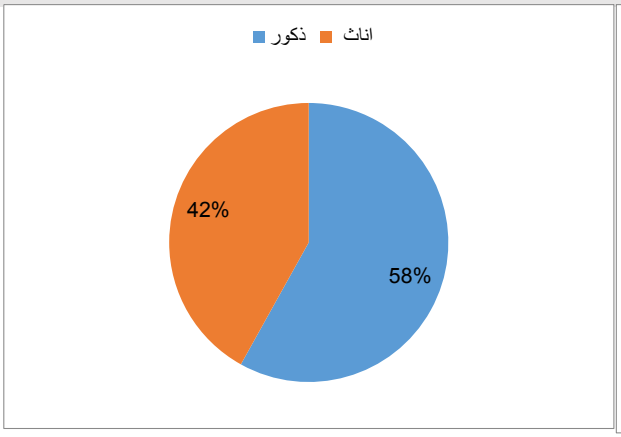
يوضح الشكل (7) تدني الوضع التطعيمي في محافظات ,صعدة ,الجوف , ابين , لحج , صنعاء , امانة العاصمة , ذمار , شبوة , البيضاء , الحديدة , الضالع ,عدن و سيئون في حين كانت افضل المحافظات في الوضع التطعيمي في محافظات المحويت , مارب , المهرة وسقطرة

شكل (8)يوضح نتائج المتابعات الستينية لحالات الشلل الرخو الحاد الى الاسبوع 48للعام 2022



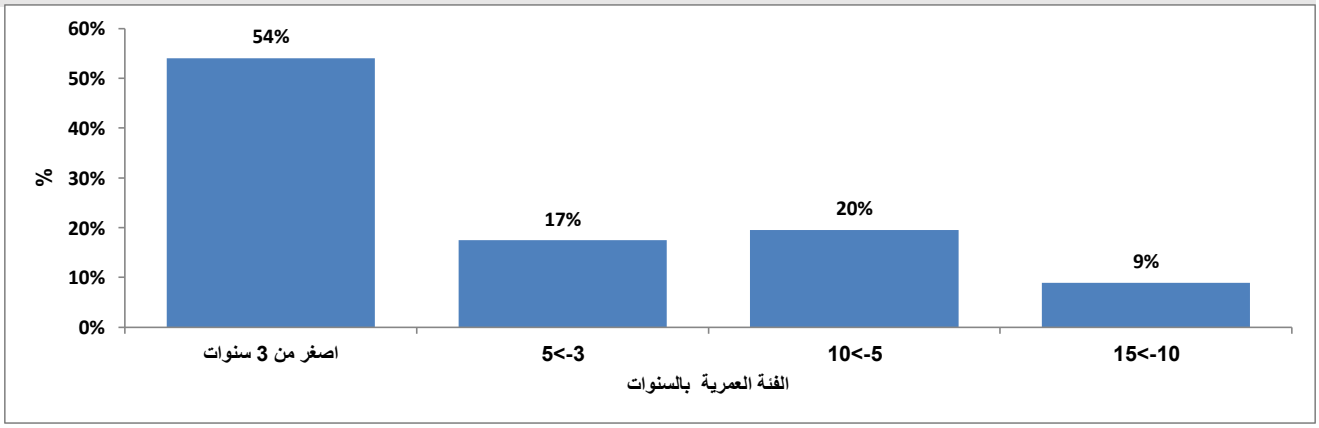
يظهر الشكل (8) انه تم اجراء 1006 متابعة ستينية بنسبة 96% من اجمالي الحالات المطلوب متابعتها حتى الاسبوع 48 - 2022م منها 411 (41%) حالة فيها اثر متبقي و 71 (7%) توفيت او فقت و524 (52%) لا يوجد فيها اثر

شكل (9)يوضح توزع حالات الشلل الرخو الحاد حسب الجنس الاسبوع 48-2022



الشكل(9) يوضح ان 58% من حالات الشلل الرخو الحاد هي من الذكور

شكل (10) نسبة حالات الشلل الرخو الحاد بحسب الفئات العمرية حتى الاسبوع 48-2022م



البرنامج الوطني لترصد الشلل الرخو الحاد

Epidemiology and prevention of Whooping cough

Dr. Nora Al-Awadhy

Whooping cough

Definition and Causes:

- ✓ **A** childhood infection which is highly contagious caused by a Bordetella Pertussis(viral infection)**.**
- ✓ **Mode of infection.**

Direct and indirect

- coughs, sneezes or talks, infected droplets spray into the air, where other people can inhale them.

Whooping cough

It belong to Myxo-group of air born viruses .

✓ **Sources of infections**

- Human is the only source.
- No carriers .

Epidemiology:

✓ Both sexes.

✓ Infants and children under 5 years.

✓ Active immunity(solid) after infection.

✓ More prevalent during March and April in all seasons.

✓ Place: worldwide (high prevalence in tropical countries in compared to cold countries).

Whooping cough

- The infection occurs in a sequence of stages
- **Infection and incubation.**
- **incubation period** 1-2 weeks.
- **inflammatory period (3 stages)**.
- Catarrhal stage (10 days)(rhinitis, sneezing, dry cough)
- Paroxysmal stage(2-4 weeks) (paroxysmal attack of coughing, whoop(Forceful inspiratory gasp), expectoration of mucus and vomiting) .
- Convalescence stage where whooping and vomiting decrease until it stops(cough may continued for weeks).

Whooping cough

Complications

Infants < 6 months have high mortality and morbidity

- Bronchitis
- Bronchopneumonia 25%(90% mortality rate)
- Atelectasis due to mucous plug then later Bronchiactasis.
- Sub conjunctival hemorrhages, Epistaxis rarely.
- Hemoptesis
- Convulsions 4%
- Cerebral hemorrhage----death.

Whooping cough

Diagnosis—

Clinically

- characteristic paroxysmal attack of coughing followed by whoop with expectoration of clear mucous and vomiting.
- History of incomplete vaccination or no vaccination.

investigation

- Leukocytosis(lymphocytosis),Nasopharyngeal swab (isolated in 90% of cases), chest Xrays(to exclude complication).

Whooping cough

Prevention- no maternal acquired immunity for infants although second attack may occur.

General

- ✓ Oxygen and gentle suctioning.
- ✓ Rest
- ✓ Good nutrition and hydration
- ✓ Prevention of Overcrowding,
- ✓ Proper ventilation

Specific

- ✓ The Centers for Disease Control and Prevention recommends that children and adults receive the vaccine DPT, DaPT, PT.
- ✓ Passive immunization by globulin.
- ✓ **Infants < 3 months must be admitted (risk of apnea and cyanotic spells).**
- ✓ **Treatment of complication.**

Whooping cough

Control

For cases – early diagnosis

- ✓ Isolation at home for >=5 days after initiation of antibiotic therapy.
- ✓ Symptomatic treatment
- ✓ Antibiotics as erythromycin 40-50 mg/kg/24 hours OID for 14 days, clarithromycin 15-20 mg/kg/day Bid for 7 days.
- ✓ Disinfection of the discharge from nose and throat.

For contacts

- Listing all contacts to identify the susceptible and non susceptible group.
- Surveillance for 2 weeks and examination for detection of missed and atypical cases..
- Booster dose of DPT for children below 4 years.

THANKS

Soil Transmitted Helminthiasis

Dr. Aisha Khaled Al-da’eri

Introduction

** Soil-transmitted helminth (STHs) infections refer to a group of parasitic diseases in humans caused by:

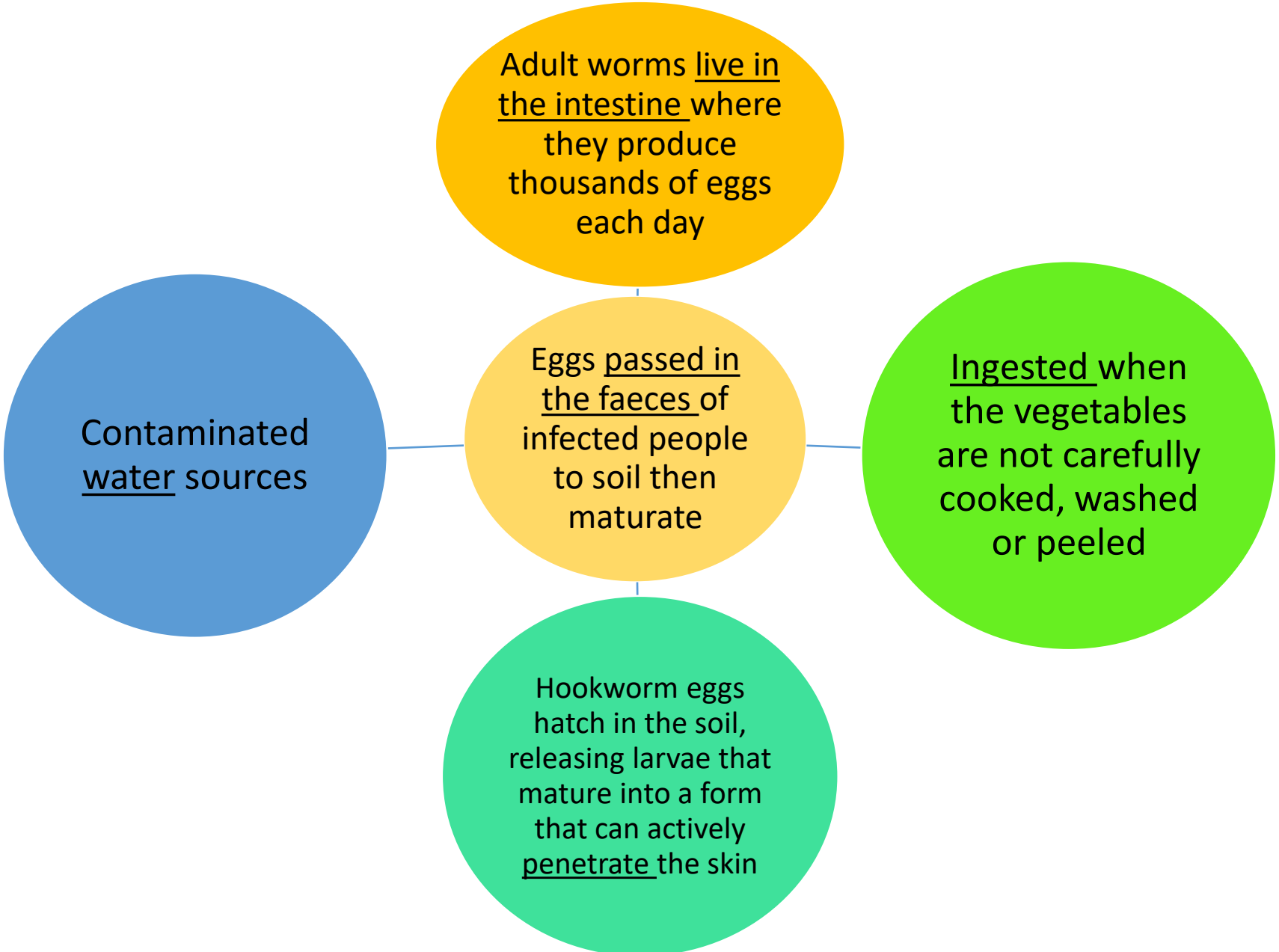
1-Intestinal roundworms (ascariasis).
2-Hookworms (~~Necator americanus and~~ Ancylostoma duodenale)
3-Whipworm (Trichuris trichiura).

More than **1.5 billion people, or about **24 %**of the world's population are infected.

**Infections are widely distributed in tropical and subtropical areas, with the greatest numbers occurring in sub-Saharan Africa, the Americas, China and east Asia .

خريطة للتوزيع

Mode of Transmission


Adult worms live in the intestine where they produce thousands of eggs each day
Contaminated water sources
Eggs passed in the faeces of infected people to soil then maturate
Ingested when the vegetables are not carefully cooked, washed or peeled
Hookworm eggs hatch in the soil, releasing larvae that mature into a form that can actively penetrate the skin

Is direct person-to-person transmission possible?

There is no direct person-to-person transmission, or infection from fresh faeces, because eggs passed in faeces need about three weeks to mature in the soil before they become infective.

Mode of transmission

Can the human host reinfect himself?

Since these worms do not multiply in the human host, reinfection occurs only as a result of contact with infective stages in the environment

Ascariasis

- Most common helminthic infestation.

- An infection of the intestinal tract caused by the adult, Ascaris lumbricoides and clinically manifested by vague symptoms of **nausea**, **abdominal pain and cough**. Live worms are passed in the stool or vomited. Occasionally, they may produce **intestinal obstruction** or may migrate into the peritoneal cavity.

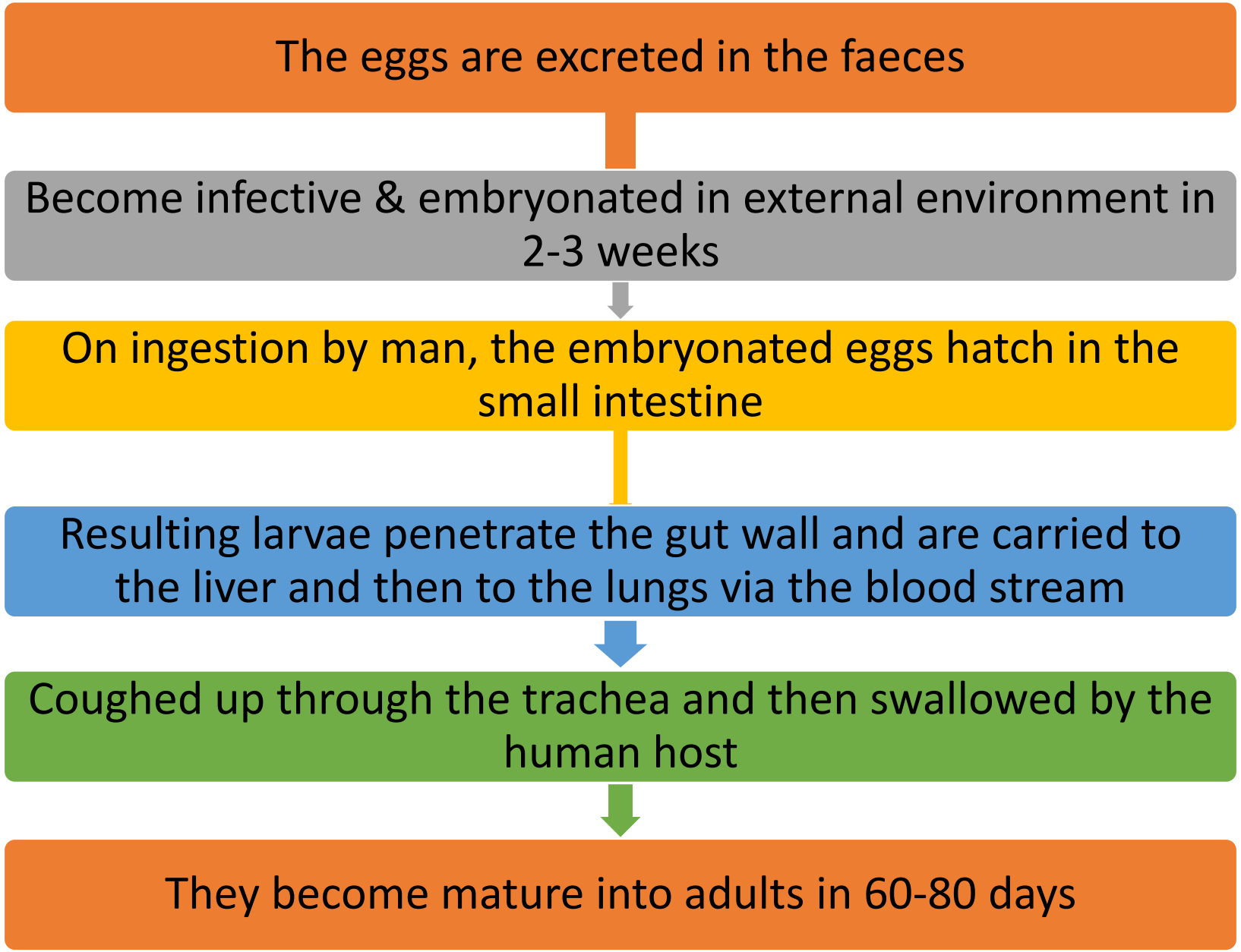
Epidemiological Features :

__AGENT**__ :

- Ascaris lumbricoides lives in the lumen of small intestine, where it moves freely. Sexes are separate.

-The female measures __**20-35**__ mm in length, and the male __**12-30**__ mm.

- Egg production is very heavy - an estimated __**240,000**__ eggs per day by each female.
- The life span of an adult is between __**6-12**__ months, maximum reported being 1.5 years.


The eggs are excreted in the faeces
Become infective & embryonated in external environment in 2-3 weeks
On ingestion by man, the embryonated eggs hatch in the small intestine
Resulting larvae penetrate the gut wall and are carried to the liver and then to the lungs via the blood stream
Coughed up through the trachea and then swallowed by the human host
They become mature into adults in 60-80 days

Epidemiological Features :

**RESERVOIR OF INFECTION :

Man is the only reservoir.

**INFECTIVE MATERIAL :

Faeces containing the fertilized eggs.

**HOST :

Infection rates are high in children.

Epidemiological Features :

**ENVIRONMENT :

-Ascaris is a "soil-transmitted" helminth. The eggs remain viable in the soil for months or years under favorable conditions.

-Clay soils are most favorable for the development of ascariasis eggs, in contrast to moist porous soils for those of hookworms

**Incubation period:

- 18 days to several weeks.

** The WHO definition of heavy infection of roundworm is ~ 50,000

eggs per gram of faeces.

Symptoms:

The symptoms are related to the number of the worms harbored. People with light infection usually have no symptoms. Heavier infections can cause a range of symptoms:

- Diarrhea, abdominal pain; general malaise, weakness, impaired cognitive and physical development.

- The larvae migration may cause fever, cough, sputum formation, asthma, skin rash, eosinophilia. The adult roundworm aggregate masses can cause volvulus, intestinal obstruction or intussusception; and wandering worm can cause bowel perforation in the ileocecal region, blocking of common bile duct or may come out with vomit

- Roundworms rob man of his food and may possibly compete for vitamin A in the intestine. They contribute to malnutrition especially in children who may show growth retardation.

HOOKWORM INFECTION

Ancylostoma duodenale and Necator americanus are the main nematodes causing hookworm infection in man.

Epidemiological Features :

**AGENT :

- Adult worms live in the small intestine, mainly jejunum where they attach themselves to the villi.

- Males measure 8 to 11 mm, and females 10 to 13 mm in length with dorsally curved anterior end.

- One female A duodenale produces about **10,000-30,000** eggs and one female N. americanus about **5,000-10,000** eggs per day.

- Adult A duodena/e and N. american us are believed to be capable of surviving for an average about **1 and 4 years**, respectively

deposited on warm, moist soil, a larva rapidly develops in the egg and hatches after 1 to 2 days.

becomes a skin-penetrating third stage infective larva within 5 to 10 days

They can survive in shaded, moist soil for up to one month

Resulting larvae penetrate the skin

Once inside the body, they migrate via lymphatics and blood stream to the lungs

coughed up and swallowed to reach the small intestine, where they become sexually mature

Epidemiological Features :

**RESERVOIR:

Man is the only important reservoir of human hookworm infection.

**INFECTIVE MATERIAL :

Faeces containing the ova of hookworms.

Epidemiological Features :

**Host factors:

(a) AGE AND SEX: All ages and both sexes are susceptible to infection., the highest incidence is found in the age group, 15 to 25 years.

(b) NUTRITION : Studies indicate that malnutrition is a predisposing factor

(c) OCCUPATION : It is to be expected that hookworm infection will have a higher prevalence in agricultural than in town workers, and in many tropical countries, it is an occupational disease of the farming community.

Epidemiological Features :

**Environmental factors :

Hookworm larvae live in the upper half-inch (1.2 cm.) of the soil.

(a) SOIL : sandy soil is more favorable than clay soil.

(b) TEMPERATURE : A temperature of 24 to 32 deg. Larvae are killed at 45 to 50 deg.

(c) OXYGEN : This is required for the growth and development of the larvae.

(d) MOISTURE : Moisture is necessary for survival; dryness is rapidly fatal.

(e) RAINFALL: More important than the total annual rainfall is the number of rainy days spread out evenly throughout the year to keep up the moisture content of the soil. Flooding is an unfavorable factor.

(f) SHADE : Direct sunlight kills the larvae whereas shade protects them.

(g) HUMAN HABITS : These include indiscriminate defecation, using the same places for defecation, going barefoot, farming practices using untreated sewage, children wading in the infected mud with bare-feet and hands. These habits are compounded by social factors such as illiteracy, ignorance and low standard of living.

Epidemiological Features :

**Incubation period (prepatent period) :

A duodenale is unpredictable, ranging from 5 weeks to 9 months. This is because the invading larva of A. duodenale is capable of remaining arrested or dormant in the tissues of the host for as long as 9 months and then again resume development and migration.

Effects of the disease of hookworm infestation :

{a) INDIVIDUAL :

1-Chronic blood loss and depletion of body's iron stores leading to iron-deficiency anaemia.

2-Retarded physical growth and development in children

3- Increase morbidity, low birth weight babies, abortion, stillbirths and impaired lactation in mothers

4-Diminished capacity for sustained hard work for adults in general .

5- Hookworm infection also causes a loss of blood plasma into the small intestine which can lead to hypoalbuminaemia in some subjects.

{b) COMMUNITY :

1- Exerts a significant and harmful effect on various aspects of the economy and quality of life of a community, especially in three major areas. These are development; work productivity, and medical care costs.

WHIPWORM

Whipworm is the third most common soil-transmitted helminthiasis in the humans. According to current estimate, nearly 800 million people are infected, and majority of cases are children 4-10 years age. Heavy infection could lead to acute symptoms such as diarrhoea and anaemia, and chronic symptoms such as growth retardation and impaired cognitive development.

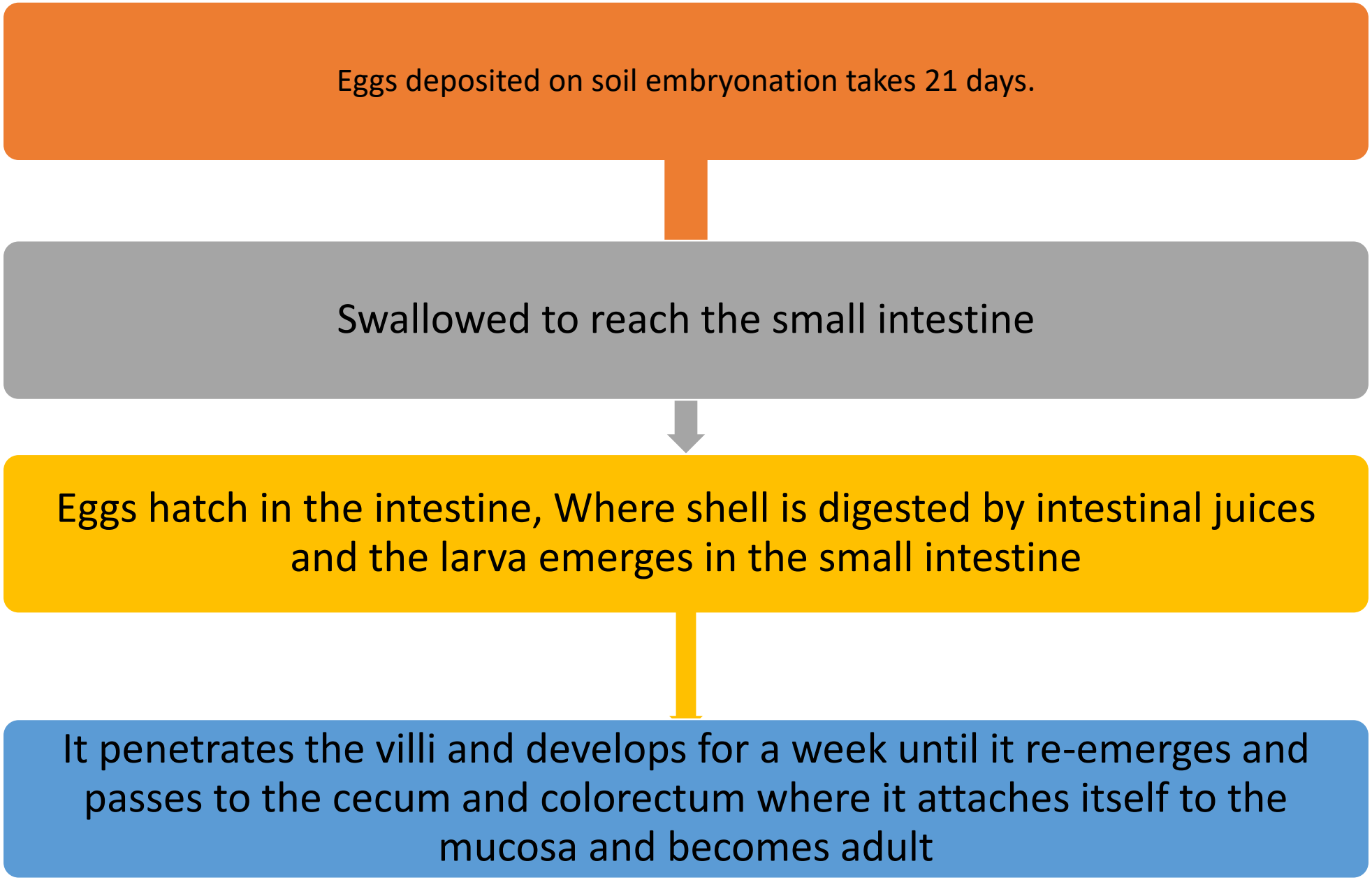
Epidemiological Features :

- Agent Factor Whipworms live in the large intestine.

- Male worm measures about 30-45 mm long while female is about 30-35 mm long.

- Female worm produces about 200-10,000 eggs per day for more than 5 years.

- Incubation period from ingestion of egg to appearance of egg in stool is 60-90 days.


Eggs deposited on soil embryonation takes 21 days.
Swallowed to reach the small intestine
Eggs hatch in the intestine, Where shell is digested by intestinal juices and the larva emerges in the small intestine
It penetrates the villi and develops for a week until it re-emerges and passes to the cecum and colorectum where it attaches itself to the mucosa and becomes adult

Effect of Whipworm infection :

'Majority of infections are mild or asymptomatic.

1-It causes epigastric pain, nausea, vomiting, distension flatulence, weight loss.

2-Moderate infection causes growth deficit and anemia.

3-Severe infection causes severe chronic diarrhea or dysentery with blood and mucous in stool, dehydration, rectal prolapse, colonic· obstruction, hypoproteinemia, chronic iron deficiency anemia .

PREVENTION AND CONTROL OF SOIL-TRANSMITTED HELMINTHS:

Primary prevention Methods:

1-Sanitary disposal of human excreta

2- Provision of safe drinking water.

3- Food hygiene habits.

4-Health education of the community in the use of sanitary latrines, personal hygiene and changing behavioral patterns.

5-Measures of personal protection such as wearing protective footwear

N.B: Prevention, to be effective, must take into consideration the life cycle of the parasite and the peculiar ecological, social and cultural circumstances that prevail in a community.

PREVENTION AND CONTROL OF SOIL-TRANSMITTED HELMINTHS:

Secondary prevention :

(a) Albendazole : The usual dose for adults and children over 2 years is 400 mg as a single dose. Contraindicated in children below 2 years and in pregnancy.

(b) Mebendazole : The usual dose is 100 mg twice daily for 3 days for all ages above 2 years.

(c) Levamisole : For many it is now the drug of choice. A single oral dose of 2.5 mg/kg of body weight (maximum 150 mg) has been recommended. There are usually no side effects. It has been used successfully in the mass treatment of ascariasis.

(d) Pyrantel: Effective in a single dose of 10 mg/ kg of body Weight with a maximum of 1g.

Secondary prevention :

Treatment of anemia and other complications.

1-Anaemia should be treated with iron and folic acid.

2-Patient should be treated for hypoproteinemia and hyper eosinophilia.

Preventive chemotherapy :

(1) The WHO strategy for control of soil-transmitted helminth infection is to control morbidity through periodic treatment of at-risk people living ·

(2) In endemic areas. The people at risk are : (a) preschool-aged children; (b) school aged children; {c) women of childbearing age including pregnant women in second and third trimesters and breastfeeding mothers and adults in certain high-risk occupations, such as tea-pickers or miners.

(3) WHO recommends periodic deworming without previous individual diagnosis to all at-risk people living in endemic areas.

(4) Treatment should be given once a year when prevalence of soil-transmitted helminth infection in the community is over 20 per cent and twice a year if the prevalence is over 50 per cent.

(5) The recommended medicines are albendazole { 400 mg) and mebendazole {500 mg}. They are effective and can be easily administered by non-medical persons.

(6) The global target is to eliminate morbidity due to soil transmitted helminthiasis in children by year 2020.>> not reached

Thank you,,,

H.W

What is the drug of choice for pregnant woman & children below 2 years?

Acute respiratory infection

Dr. Rasha Nujaim

About community medicine

Def.

Levels of health care

Elements of primary health care

Priorities of health problem in Yemen

Mortality rate of children under 5 years

ARI

Types of ARI:URTI

LRTI

Problem statement:

worldwide death of children every year 3.9 million

90% of the cause is pneumonia with prevalence 20-30% in the developing countries

More than 1.1 million under 5 years of age children die from pneumonia each year

Majority of death occurring in sub Saharan Africa and South Asia

Epidemiological determinants

Agents factors

Host factors

Risk factors

Mode of transmission

Control of ARI

Clinical assessment

Physical examination

Classification of illness

- Very sever disease
- Sever pneumonia
- Pneumonia
- No pneumonia

prevention

Immunization

IMCI

GAPPD

THANK YOU

Acute diarrheal disease

DR. RASHA NUJAIM

INTRODUCTION

Clinical types :

Acute watery diarrhea

Acute bloody diarrhea

Persistent diarrhea

Problem statement:

Killed more than 1600 children under 5 years of age every day in 2012

In Yemen it is 1st health problem

Epidemiological determinants

Agent factors

Virus

Bacterial

Parasites

Reservoir of infection

Host factors

Environmental factors

Mode of transmission

Control of diarrhea

- Appropriate clinical management

-ORS

- Intravenous rehydration

Maintenance therapy

chemotherapy

-feeding

Preventive strategies

Sanitation

Health education

immunization

Thank you